The American Journal of Surgery

EDITED BY

Thurston Scott Welton, M.D., F.A.C.S.

GENITOURINARY SURGERY · GYNECOLOGY

GASTROINTESTINAL SURGERY · UROLOGY

ORTHOPEDIC SURGERY · PROCTOLOGY

GENERAL SURGERY · NEW INSTRUMENTS

August 1940

THE AMERICAN JOURNAL OF SURGERY, Inc.

49 WEST FORTY-FIFTH STREET NEW YORK, N. Y.

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The Rationale of Suture Use

BRIEF DISCUSSIONS BASED ON REPORTS OF CURRENT RESEARCH AND CLINICAL OBSERVATION

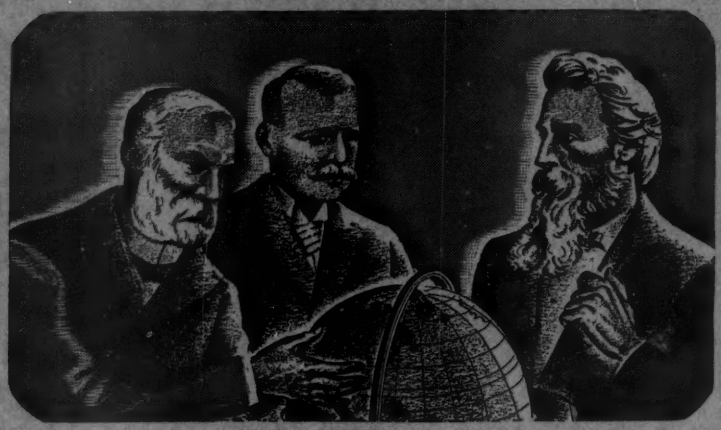
AT NO PERIOD in surgical history has interest in the suture and various phases of wound healing been more active than at present. Through the discussion which it has promoted there is, however, reiteration of the theme that a particular type or variety of material will yield superior results under all circumstances. Nothing could be further from the truth. Uncertain, or poor results are obtained with all, and excellent results with many—*depending entirely upon the manner and circumstances in which they are used.*

Analysis of more optimistic conclusions has invariably disclosed failure to consider important contributing factors. Comparisons are made between materials without regard for marked differences in their sizes. Long-term statistics and experiences from other eras are cited without consideration of later changes in technic or improvement in the materials themselves. The effect of trauma, tension, incomplete hemostasis, and various pathologic conditions are often not accorded full significance.

This reflects no lack of sincerity on the part of the investigators. Rather it emphasizes the extreme difficulty of maintaining detailed charts and uniform control over all things in routine operative procedure, coupled with the virtual impossibility of disassociating the work from a particular set of circumstances which may influence the pattern of results.

Unfortunately, the surgeon seeking to interpret the data for application in his own work is confronted with the same difficulties and this undoubtedly explains the wide variance in clinical experience.

It is believed, therefore, that a correlation of reports from various sources, interpreted in the light of long experience in this type of investigation and supported by carefully charted experimental work, would help to clear up existing confusion. With this thought, Davis & Geck, in collaboration with recognized authorities, is preparing a series of brief discussions which will be presented in these pages from time to time.



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NOTE: In agreement with the new U. S. P. nomenclature, catgut products formerly known as Plain, 10-day, 20-day, and 40-day, are now designated as

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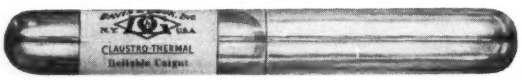

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954	Half-curved	20″	000, 00, 0	3.00
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				NET, PER DOZ. BOXES
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			000	11.64
			00	12.60
			0	14.40

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40341	Straight	28″	4-0	3.60
40344	Half-circle	28″	4-0	4.20
40245	None	54″	4-0	3.60

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50445	None	54″	5-0	3.60
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360	Horsehair	6 x 28″	00
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400	Black silkworm gut	6 x 14″	00, 0, 1
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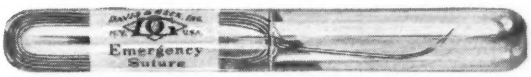

WITH HALF-CURVED NEEDLES

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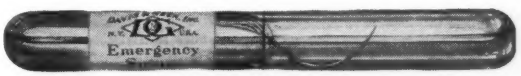
904	Plain—type A catgut	18″	00 to 3
914	Mild chromic—type B	18″	00 to 3
924	Medium chromic—type C	18″	00 to 3
954	Kal-dermic	20″	000, 00, 0
964	Horsehair	2 x 28″	00
974	White silkworm gut	2 x 14″	00, 0
984	White twisted silk	20″	000, 0, 2
986	Anacap silk	20″	000, 0, 2
900	Assorted: Catgut, Silk, and Kal-dermic		



WITH ⅜-CIRCLE NEEDLES

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923	Medium chromic—type C	18″	00 to 2
953	Kal-dermic	20″	000, 00, 0
963	Horsehair	2 x 28″	00
973	White silkworm gut	2 x 14″	0
983	White twisted silk	20″	000, 0, 2
985	Anacap silk	20″	000, 0, 2
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TO surgeons of the Arabian school belongs the credit for preserving the science of surgery in the period following the decline of Greco-Roman civilization. In the use of sutures and ligatures they followed existing practices rather closely with one notable exception—the writings of Rhazes describe the closure of wounds with harp strings, made from the intestines of sheep, twisted and dried in the sun, constituting the earliest record of the use of catgut as a suture material.

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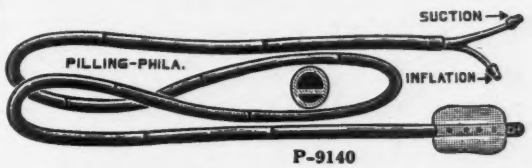

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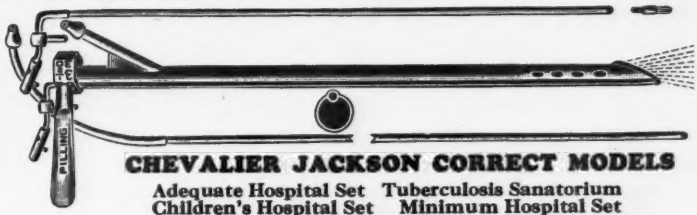

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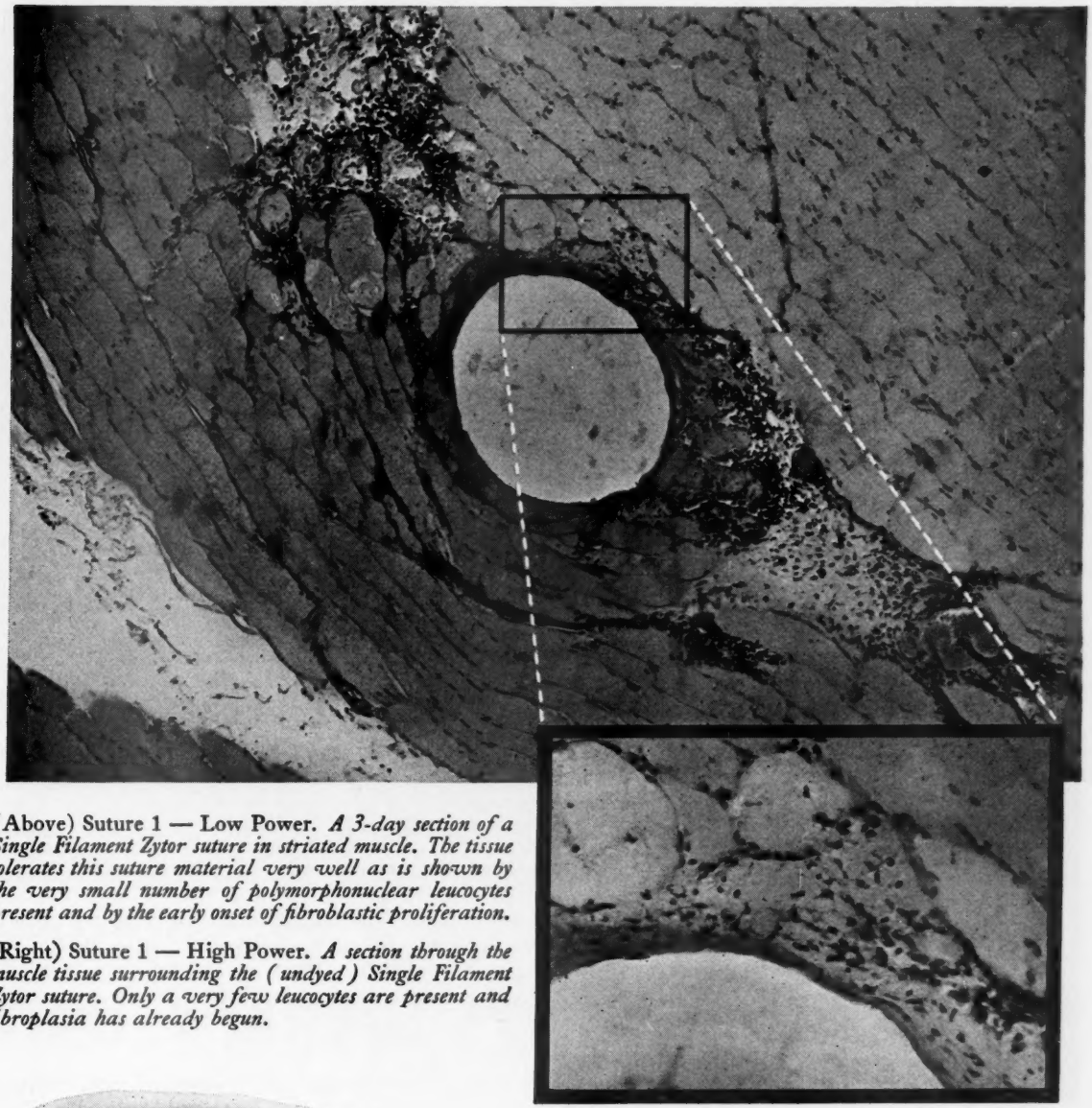
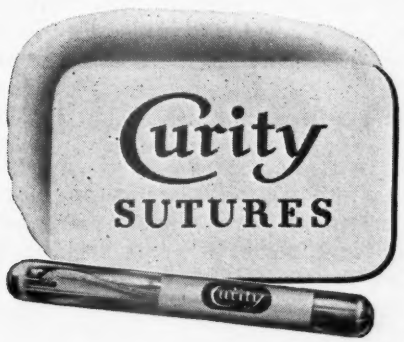


(Above) Suture 1 — Low Power. *A 3-day section of a Single Filament Zytor suture in striated muscle. The tissue tolerates this suture material very well as is shown by the very small number of polymorphonuclear leucocytes present and by the early onset of fibroblastic proliferation.*

(Right) Suture 1 — High Power. *A section through the muscle tissue surrounding the (undyed) Single Filament Zytor suture. Only a very few leucocytes are present and fibroplasia has already begun.*



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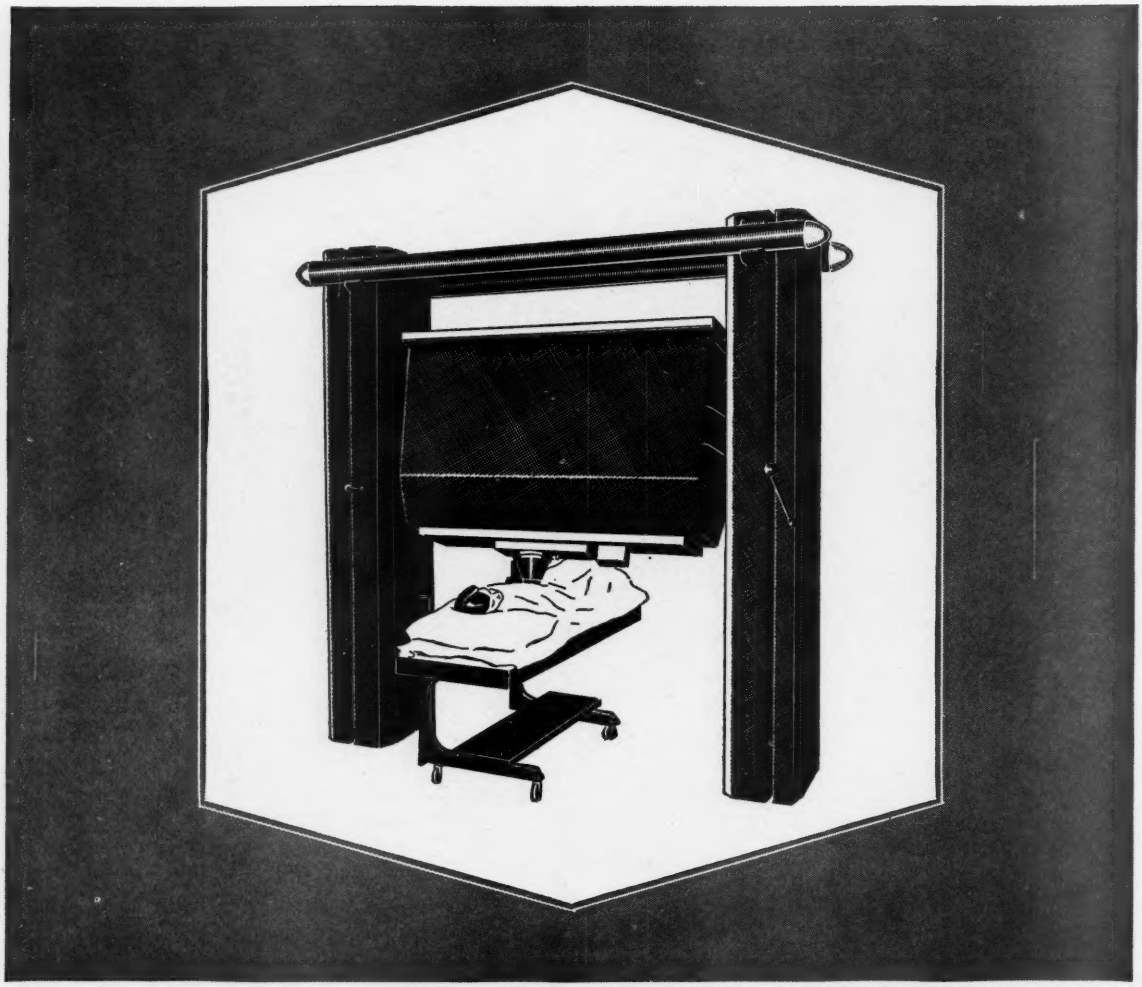

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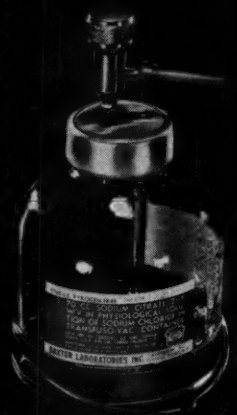

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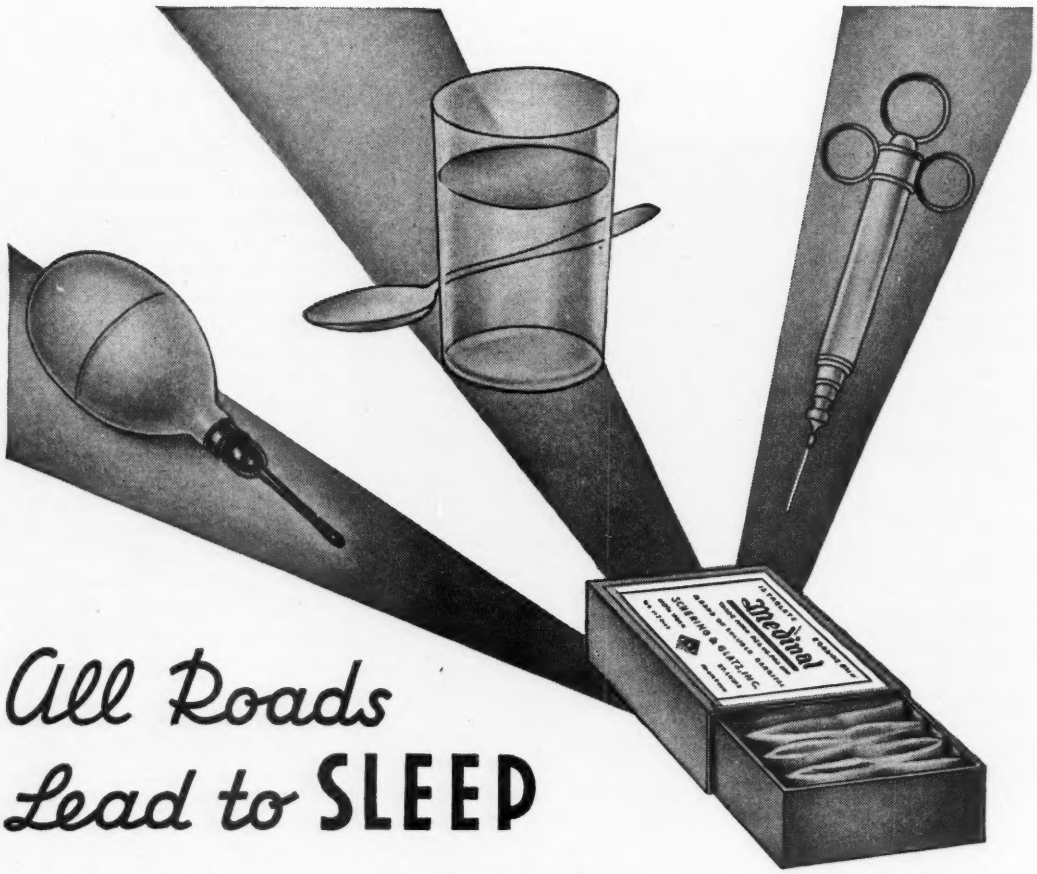

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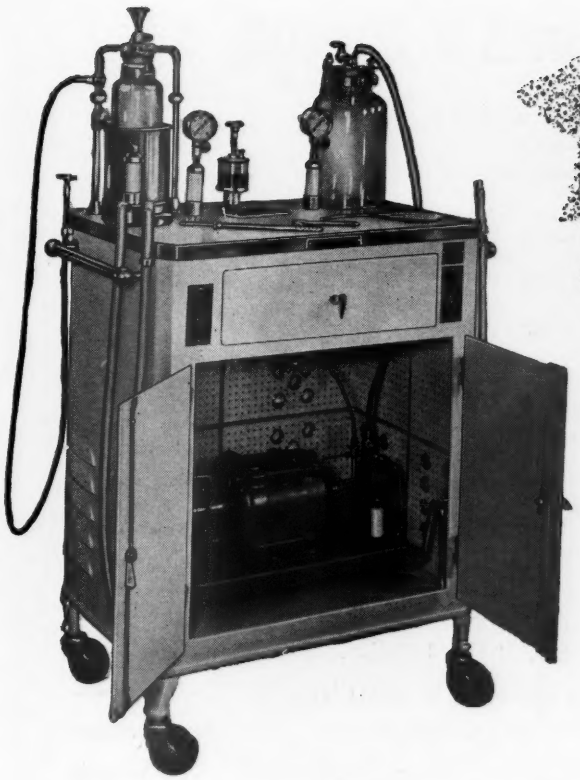

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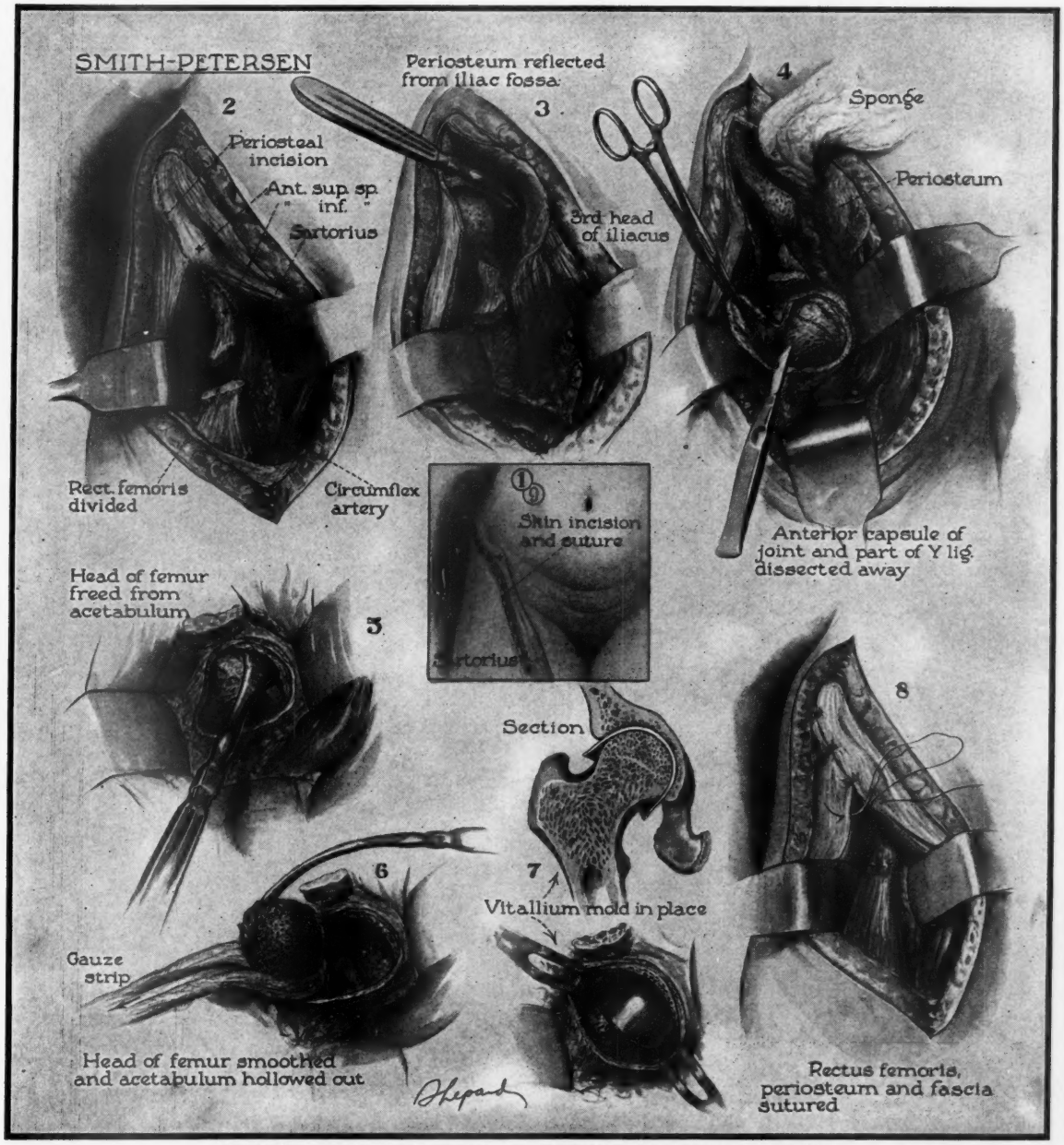

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CORRECTION—In an obvious error, our text matter under the July Operative Procedure, "Gastrostomy," recommended Arterial Sutures for the operation. The *correct* material, of course, is Surgical Gut.

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NEW SERIES, VOL. XLIX AUGUST, 1940 NO. 2

The American Journal of Surgery

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A PRACTICAL JOURNAL BUILT ON MERIT

EDITORIAL

WHEN?

PHYSICIANS are human beings and citizens of our United States and most of them have many questions that persist in the front of their minds—questions that call for answers.

We know only what our leaders through the press tell us. We know England and her navy is the last of the Allied defense; and we know, as we write this, that an attack on England is expected any minute. Should England be invaded or fall apart, as France did, will we be put on the spot? Is it true we are far from prepared? Could a strong foreign power with a navy prove an actual menace? When we are told we are in danger, does it mean *now, two months* from now or *three years* from now? If the real danger is right around the corner, must we wait ten months to begin the production of air-planes, two or three years to properly train twenty-five thousand aviators, four or five years to complete present navy contracts? Is it true it will take a couple of years to manufacture sufficient rifles to supply an army of less than half a million men? And how long will it take to make sufficient anti-aircraft guns, large tanks and the thousand other things that spell preparedness?

Is it true that early in the war Canada found herself in a bottle-neck regarding aviation because of insufficient numbers of physicians who knew the medical technique of examining applicants for the flying service? If true, how about this country? Have we enough physicians who have been trained in this work? If not, why not? War calls for expert knowledge in many of the medical and surgical branches. This was learned in the last great war. Are the medical schools making plans for undergraduate and postgraduate instruction in these specialities and fields? To date we have not heard of any move in this direction. On one hand we are told the government has every physician classified and indexed and at a moment's notice can assign him for the role he is to play; on the other hand we hear the rumor that the American Medical Association will be the headquarters and it will notify the various State organizations and through them the County societies which will fill the necessary quota of physicians. What are the true facts?

If we are in peril, why do we not take off our coats, roll up our sleeves and get to work? This means the ditch digger, the mechanic, the specialist and the doctor of medicine. Why at this time a six-hour day and a five-day week? France had that and see where she got the axe.

When are we going to stop talking and get to work—the day after a war has formally been declared? France and Eng-

land, poorly led and ill prepared turned to us and cried for help. Should our turn come, to whom can we apply? What nation deep in its heart loves us sufficiently to make the supreme sacrifice for us? You know the answer . . . but when will we get going . . . when?

T. S. W.

JOURNAL OF THE NATIONAL CANCER INSTITUTE

THE National Cancer Institute, of the National Institute of Health, United States Public Health Service, announces the forthcoming issue of *The Journal of the National Cancer Institute*, the official organ of the Institute.

The new journal, to be issued bimonthly, will be of the scientific type of periodical and will contain articles by the members of the staff on the various lines of cancer research work carried on by the Institute.

The first issue will contain papers on the Federal cancer control program, approaches to cancer research, effect of various hydrocarbons in producing tumors in mice and studies on normal and cancerous tissues.

Copies of the journal will be sold to applicants by the Superintendent of Documents, Government Printing Office, Washington, D. C., who will also handle all annual paid subscriptions. The journal will be distributed free to a limited number of medical schools, to workers in the field of cancer research, research institutes interested in cancer, to a limited number of surgeons, as well as to certain Government depositories, and to journals making suitable exchanges.

ORIGINAL ARTICLES

CERVICAL SPINE TRAUMA ASSOCIATED WITH INJURY OF THE HEAD AND SHOULDER GIRDLE: COLEMAN'S SYNDROME*

J. M. MEREDITH, M.D. AND EVERETT GRANTHAM, M.D.

Assistant Clinical Professor of Neurosurgery, University of Virginia School of Medicine

UNIVERSITY, VIRGINIA

University of Louisville School of Medicine

LOUISVILLE, KENTUCKY

A NUMBER of years ago it was emphasized by Dr. C. C. Coleman that if the head and a shoulder are traumatized simultaneously, the cervical spine must often sustain forces acting in opposite directions and is very likely to be injured where the two forces converge. A natural result of this observation was greater vigilance in searching for a possible injury of the cervical spine when the examination disclosed a combined injury to the head and shoulder girdle. This combination of injuries was found so frequently in the Clinics that it has come to be known among the resident staff as Coleman's syndrome.

It is of great importance that a systematic examination of the entire body, as soon as the general condition of the patient permits, be made routinely when a head injury is present. Disturbance of consciousness from a head injury often makes it difficult to recognize important associated injuries in other regions of the body. The delayed recognition of injuries of the chest, spine and long bones may result in serious consequences to patients with head trauma, which latter lesion, in many cases, may not be severe enough to threaten the patient's life.

The associated cervical spine injury is especially apt to be overlooked: (1) when there is no evidence of trauma to the spinal cord, such as would be indicated by paralysis or bladder disturbance; (2) in an unconscious patient who has obvious signs of a shoulder girdle injury, such as a fractured clavicle, but is unable to coöperate in a general examination; (3) in a patient with bloody spinal fluid from intracranial injury. In the last group of cases the rigidity of a broken neck is often erroneously attributed to subarachnoid hemorrhage from the head trauma until *x*-ray examination of the cervical spine shows the true cause.

The importance of this combination of injuries should be emphasized. Injury to the cervical spine should be suspected at the scene of the accident, if a combined head and shoulder injury is discovered by the physician, so that the cervical spine may be immobilized en route to the hospital. Furthermore, at the hospital, similar precautions should be observed immediately for the protection of the cervical spine until clinical and Roentgen examinations, including a film through the open mouth to permit inspection of the odontoid process of the axis and the atlas-axis relationship, have definitely excluded cervical spine injury. The associated head injury is of no specific type. It may be a simple

* From the Departments of Neurological Surgery, University of Virginia, and The Medical College of Virginia, Dr. C. C. Coleman, Director.

Presented in the Symposium on Neurosurgery, Spring Post-Graduate Clinic, Medical College of Virginia, April 28, 1939.

concussion with or without a skull fracture. The shoulder girdle injury may be a fracture of the clavicle, acromioclavicular

Fig. 1. Case 1. Complete fracture through the base of the odontoid process (arrow) which is slightly separated from the body of the axis (open mouth view).

separation, fracture of the scapula, upper end of the humerus, or a severe contusion of the shoulder. When one of these injuries to the shoulder girdle or upper humerus is found combined with head trauma, *x*-ray examination of the cervical spine should be done as soon as the patient's general condition permits.

The following six cases, chosen from a large number of similar ones treated in the Departments of Neurological Surgery at the University of Virginia and at the Medical College of Virginia during the last three years, emphasize the salient features of the syndrome:

Case 1. A white male, 23 years of age, was admitted to the University of Virginia Hospital, September 22, 1938 and discharged improved five days later.

On the day of admission he had fallen from a tree and was unconscious for a few minutes. He also had pain in the right shoulder and in the upper cervical spine. The patient was conscious and rational and there were no signs of spinal cord injury. An acromioclavicular strain on the right side was diagnosed by the Orthopedic Department. Skull plates showed no fracture. *X*-ray of the cervical spine demonstrated a complete fracture through the base of the odontoid process which was slightly separated from the body of the axis. The neck was immobilized in a Thomas collar and covered with a plaster cast.*

Comment. This case demonstrated the value of the open mouth *x*-ray plate in investigation of cervical spine injuries; the ordinary anteroposterior and lateral views disclosed no abnormality. Immobilization must be maintained for several months as union of an odontoid fracture is usually fibrous rather than a true bony repair.

Case 11. A white male, 28 years of age, was admitted to the Medical College of Virginia Hospital March 28, 1936 and discharged improved April 13, 1936.

The patient was brought to the emergency room immediately after an automobile accident in which he had been rendered unconscious for ten minutes. There was pain in the left shoulder and swelling in the neck posteriorly.

X-ray examination of the skull was negative for fracture. There was a comminuted fracture of the greater tuberosity of the left humerus. Films of the cervical spine disclosed comminuted fractures of the spinous processes of the second and third cervical vertebrae.

The patient was discharged sixteen days after admission in good condition (except for aphasia due to contusion of the left cerebral hemisphere), a plaster collar having been applied to his neck.

Case 111. A white male, 53 years of age, was admitted to the University of Virginia Hospital December 11, 1937 and discharged improved January 7, 1938.

The patient was unconscious for one-half hour following an automobile accident and when he regained consciousness was unable to move the right leg. Examination disclosed a Brown-Séquard syndrome with the sensory

* The writers wish to acknowledge the cooperation of the Departments of Orthopedics and Radiology at the University of Virginia and the Medical College of Virginia.

level at T_2 dermatome on the left side. Both arms were weak when extended at the elbow against resistance. *X*-ray examination disclosed no fracture of the skull but did demonstrate a fracture of the left clavicle. Films of the cervical spine demonstrated extensive injury. A small chip of bone was detached from the left dorsal border of the body of the fourth cervical vertebra. The articular processes of the third and fourth vertebrae on the left side were crushed together, as were also the left articular processes between the fifth, sixth, and seventh vertebra. The right articular process of the sixth vertebra was displaced to the left almost 0.5 cm. and the body of the sixth cervical vertebra was crushed.

The Queckenstedt test on the day of admission showed a slight subarachnoid block; the spinal fluid was clear and colorless.

As the patient's condition was good, skull tongs* were applied on the day of admission and effectively reduced the disalignment; all weights were removed six days later. Roentgen examination showed almost complete reduction of the injury with the exception of very slight forward dislocation of the sixth on the seventh cervical vertebra. A plaster cast, applied while traction was maintained, was worn for several months.

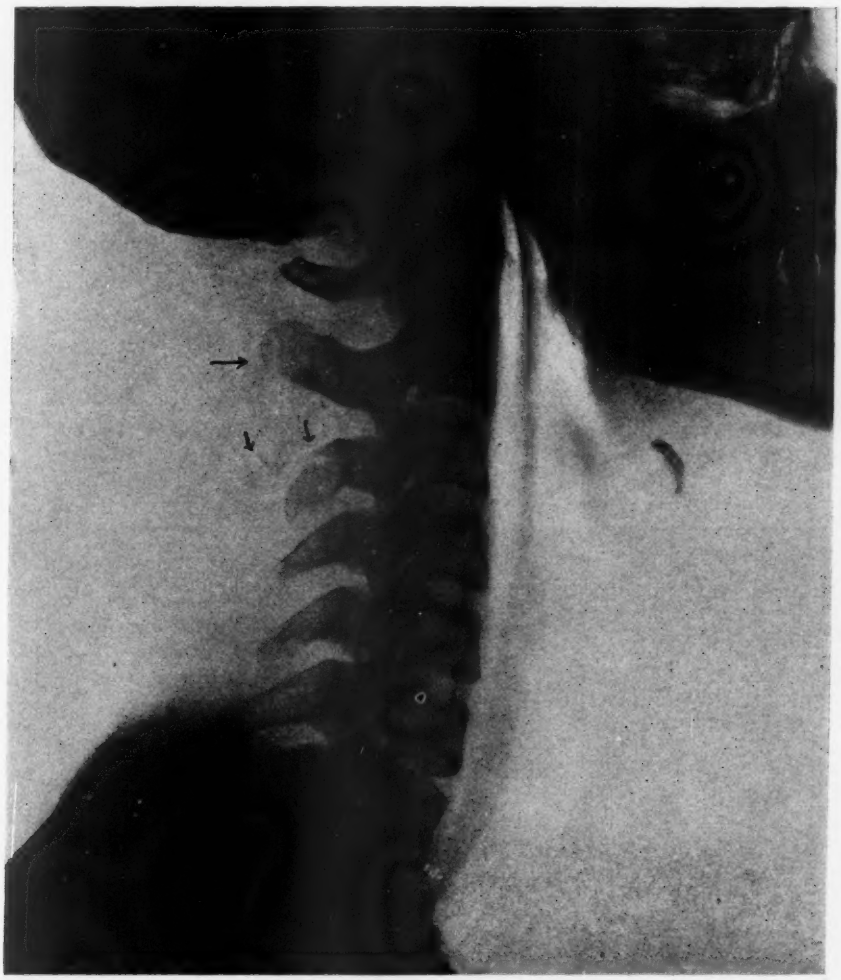

Fig. 2. Case 11. Comminuted fractures of the spinous processes of the second and third cervical vertebrae (arrows). Lateral view of cervical spine.

At the time of discharge the patient's general condition was excellent, the Brown-Séquard syndrome having almost completely

* The principle of skeletal skull traction for reduction of dislocations of the cervical spine was conceived by Dr. Coleman in 1932. An ingenious apparatus was devised by Dr. W. G. Crutchfield[1] of these Departments and has been used with great satisfaction in a large number of cases.

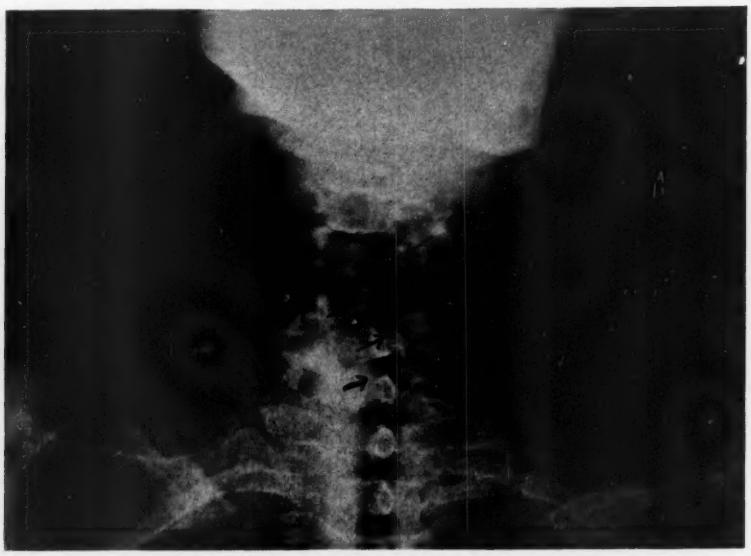

FIG. 3. Case III. Multiple fractures and crushing of greater part of the entire cervical spine (arrows). Anteroposterior view of cervical spine.

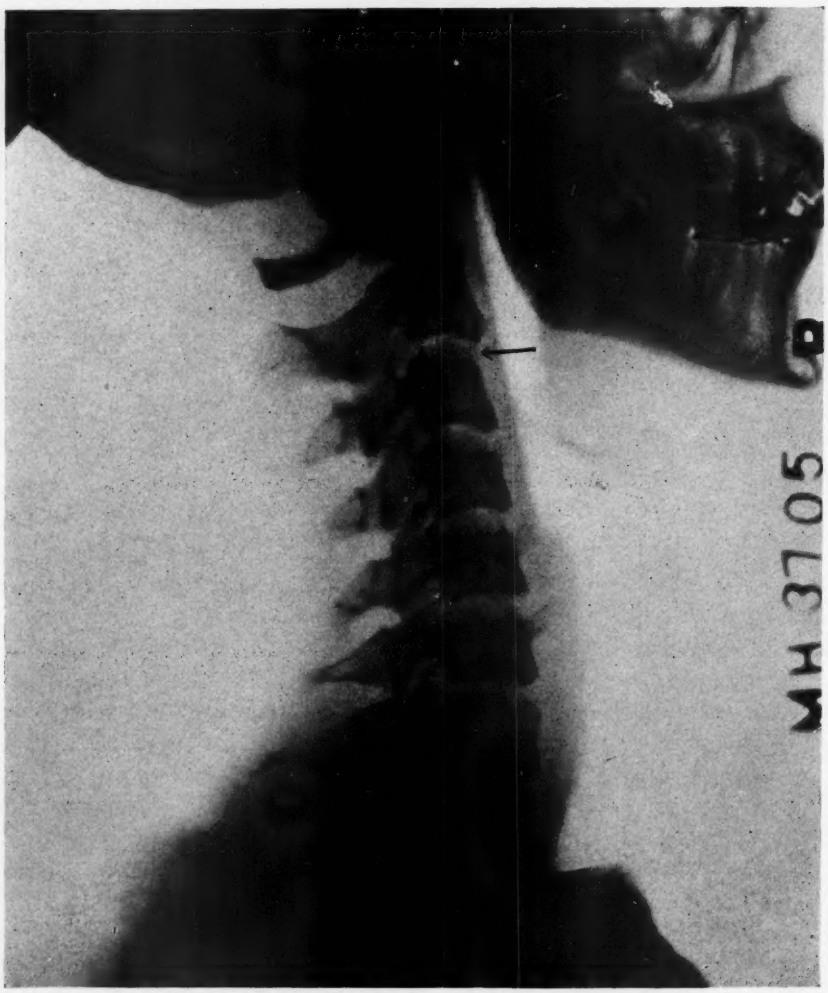

FIG. 4. Case IV. Slight dislocation of C_{2-3} vertebrae (arrow) before reduction with skull tongs traction. It is possible to have only dislocation of the cervical spine (without fracture). Lateral view of cervical spine.

disappeared. There was slight numbness of the ulnar surface of the left forearm and hand.

Comment. The skeletal traction apparatus has practically eliminated the need for laminectomy in incomplete cervical cord injuries except in those cases that have stationary or advancing neurologic signs after the dislocation has been reduced by traction and in whom the Queckenstedt test still shows an incomplete or complete subarachnoid block.[2] Cases with a Brown-Séquard syndrome (as in Case III) due to cervical cord injury and a demonstrable dislocation can almost invariably be managed solely and effectively by the skeletal (skull) traction apparatus if treated within the first few days of the injury. Open operation in cases of immediate complete cord injury, at whatever level, is usually futile; skull traction is useful in immediate complete cord lesions in the cervical region (if dislocation is present) as root pains in the arms are usually greatly relieved, although the cord injury itself remains unaffected, as a rule.

Case IV. A white male, 18 years of age, was admitted to the Medical College of Virginia Hospital September 25, 1935 and discharged improved November 9, 1935.

Fig. 5. Case V. Complete fracture through the lamina of the second cervical vertebra (arrow). A suggestive (slight) compression fracture of C_7 vertebral body was also demonstrated. Lateral view of upper cervical spine.

He was admitted a few minutes after having been injured in a motorcycle accident; he remained unconscious for twenty minutes. There was bloody spinal fluid leaking from the right ear and he complained of pain in the right shoulder. *X*-rays on the day of admission showed a fracture of the right malar bone and of the mandible; there was a distinct widening of the right acromioclavicular joint. The day after admission he complained of pain in the cervical region for the first time and *x*-ray examination disclosed anterior dislocation (0.5 cm.) of the second on the third cervical vertebra. The Queckenstedt test was normal. Skull tongs were inserted and the dislocation effectively reduced, the weights being removed one week later. A plaster cast was applied and the patient discharged in excellent condition; no signs of spinal cord injury were ever demonstrated.

Comment. It is possible to have dislocation of the cervical spine without fracture; in the dorsal-lumbar spine, fracture almost

invariably accompanies dislocation. It is most important to detect a cervical spine dislocation at the time of the injury as

Fig. 6. Case vi. An oblique fracture through the left lamina of the seventh cervical vertebra (arrow) without involvement of the neural arch. Lateral oblique view of cervical spine. The ordinary anteroposterior and lateral views of the cervical spine were normal. In suspected traumatic lesions (especially) of the cervical spine it is important to take five different views: (1) anteroposterior view through the open mouth to demonstrate atlas-axis relationships; (2) anteroposterior view of cervical spine; (3) lateral view of cervical spine, of chief value in demonstrating dislocations and spinous process fractures; (4) and (5) right and left oblique views especially to demonstrate laminal fractures.

reduction by skull tongs or by any other method is difficult after several weeks have elapsed from the time of the injury. In the meantime, the luxation may have increased with resultant cord compression.

Case v. A white female, 31 years of age, was admitted to the Medical College of Virginia Hospital August 25, 1936 and discharged improved October 26, 1936.

She was injured in an automobile accident and was still unconscious on admission, one hour or more after the injury, and remained so for several days thereafter. On admission, the diagnosis was cerebral concussion and contusion, lacerations of the scalp and the left leg. Two days later, after the patient regained consciousness, extensive Roentgen examination disclosed a linear fracture of the right frontal bone, a comminuted fracture of the left clavicle and an oblique fracture in the body of the left scapula. There was a complete fracture through the lamina of the second cervical vertebra and suggestive fractures of the left transverse process of the second cervical vertebra and of the body of the seventh cervical vertebra.

The patient remained in the hospital for two months and no treatment of the cervical spine injury—other than immobilization with a Thomas collar—was necessary. There was no spinal cord injury.

Case vi. A white male, 55 years of age, was admitted to the University of Virginia Hospital August 22, 1938 and discharged improved September 16, 1938.

On admission, on the day of injury in an automobile accident, he was in a semiconscious state, disoriented and irrational. There were multiple lacerations of the scalp and of the lower extremities. The lacerations were debrided and repaired on admission and healed per primam. Several days later, after the state of consciousness had improved, he complained of severe pain in the left shoulder which was severely contused. *X*-ray examination of the skull disclosed multiple linear fracture lines. There was little or no pain in the cervical spine although the neck was slightly spastic on forward flexion. The patient made a fairly satisfactory recovery from the head injury and was discharged twenty-five days after the injury, still mentally retarded. He returned to the hospital for examination eleven days later and (more because of our experience in previous cases of combined head-shoulder girdle injury than because of any complaint of the patient directed to the cervical spine) films disclosed an oblique fracture through the left lamina of the seventh cervical vertebra without involvement of the neural arch. A Thomas collar was applied and the patient has since had no further difficulty. There was no spinal cord injury.

Comment. These two last cases (v and vi) illustrate the importance of considering the possibility of a cervical spine injury in an *unconscious* patient with a combined

head and shoulder girdle injury. It has led us to adopt practically routine cervical spine *x*-ray examination in any such patient. The spastic neck in a patient who is unconscious from a head injury is all too readily attributed to a probable intracranial (and associated spinal) subarachnoid hemorrhage. Incidentally, Figure 6 demonstrates the value of *oblique* views of the cervical spine in traumatic cases; the usual anteroposterior and lateral views were normal in appearance in this case.

DISCUSSION

From the six cases described it may be seen that there is nothing specific about the type of cervical spine injury occurring in association with a combined head and shoulder girdle injury. The lesion may be a fracture of the odontoid process, rotary dislocation of the atlas on the axis, fracture-dislocation of one or more of the cervical vertebrae, fracture of the transverse, spinous or articular processes. Furthermore, it is important to recall that if paralysis or other neurologic signs are present in a patient with a severe head injury and a shoulder injury, such signs may just as likely be due to a *cervical cord contusion* as to a contusion of the brain. It is particularly easy to overlook a cervical spine injury if there is no neurologic involvement (which may, however, develop later if immediate treatment is not instituted) and if the patient complains a great deal of the head or shoulder injury and little or not at all of the neck injury. This occasionally occurs, as shown in Case VI; here the patient's irrational state precluded his complaining of the neck injury for several weeks—even when he became rational, he complained very little of his neck until his attention was directed to it. Such cases have led us, as stated above, to conclude that in all patients with a combined head-shoulder girdle injury it is probably best to examine routinely the cervical spine as well for possible injury. The clinical importance of early recognition of cervical spine injury and its medicolegal significance require no comment.

CONCLUSIONS

A new post-traumatic syndrome—named after the neurosurgeon who first observed and emphasized its occurrence and frequency—is described, together with six illustrative cases. This syndrome consists of a cervical spine injury as a frequent (often unsuspected) accompaniment of a combined head-shoulder girdle injury. The importance of the early recognition of the full syndrome is stressed from various standpoints.

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SECONDARY OSTEOARTHRITIS FOLLOWING FRACTURES OF THE ANKLE

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IT is generally accepted that following severe fractures of the ankle, there may be marked disability and pain which may develop into osteoarthritis (traumatic arthritis). Traumatic arthritis is an inflammatory condition of the joint. It may be the result of a single severe injury to the articular surface or repeated "microtraumata" from faulty posture, attitudes producing functional disability, and from unevenly distributed intra-articular pressures. It is manifested by changes in the bone, articular cartilage, synovia and capsule. The surrounding tissue shows chronic inflammation. Pain, swelling and limitation of motion are usually present.

FIG. 1. W. C. Case VI. Splitting fracture. Led to osteoarthritis and fusion operation fourteen months after injury.

The loss of certain normal anatomic relations of the ankle predisposes to later changes and osteoarthritis. The main anatomic changes resulting from fractures of the ankle are: (1) loss of the continuity of the weight-bearing surface of the tibia; (2) widening of the joint mortise; and (3) alteration of the weight bearing planes.

The age of the patient is definitely a controlling factor in determining the type of fracture sustained. Ankle fractures are infrequent under the third decade. Beekman[1] states that on his Children's Fracture Service at Bellevue Hospital he has never seen a Potts type of fracture. In children, the fracture is usually an inch or more

above the ankle. In Shands' series of 109 cases,[2] only nine were under 18 years of age. In seven of these nine, there was a lower tibial epiphyseal separation with no involvement of the joint. Wilson[3] reports similar findings in a series of eighty fractured ankles. After the second decade, ankle fractures become quite common and almost 30 per cent of them occur in the third decade. The malleoli may be fractured at the level of the articular cartilage of the tibia, or the tibia-fibular ligaments

A

B

Fig. 2. L. P. Case xi. Good reduction of three malleolar fractures (two malleoli plus marginal), but had definite osteoarthritis for which fusion was advised two years after injury. Causative factors were probably age of 54 and slight remaining unevenness of tibial articular surface.

may be torn and the fibula be fractured above the joint. Involvement of the articular surface of the tibia is usually seen in older patients. Mackinnon's[4] and Wilson's[3] patients with marginal fractures (posterior lip of the lower tibial articulation) averaged 44.4 years and 44.6 years respectively. It is evident that the more severe fractures are in the older group. It is well known that the restorative powers of the body decrease with age.

A B C

Fig. 3. J. A. F. Follow-up Case 11. Perfect reduction of three malleolar fractures, leaving smooth tibial articular surface. Symptom-free four and one-half years after injury, despite age of 63 years.

Shands[2] and Wilson[3] report a higher percentage of ankle fractures in women, which, we feel, is due to the increased

strain placed on the supporting structures by higher heeled shoes. This, no doubt, accounts for the greater number of marginal fractures in women. Mackinnon reported sixteen cases of marginal fractures, eleven in women. Five of the women he described as being "very heavy."

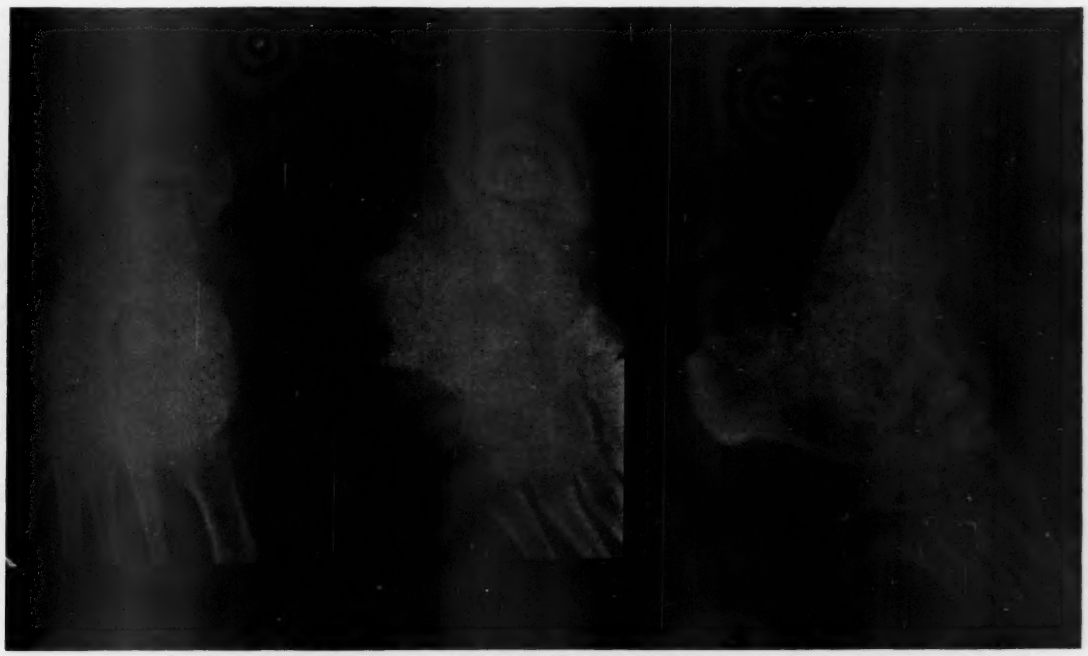

Fig. 4. S. F. Case IV. Unsatisfactory reductions of three malleolar fractures producing osteoarthritis.

The displacement of the astragalus on the tibia is seen frequently in traumatic arthritis. Shands reports it in 100 per cent of his cases of ankle fracture. If the normal relation and weight-bearing lines (as described by Skinner[5]) are not restored, the ankle is not reduced and may give trouble. Appelbach[6] reports that follow-up showed 94 per cent of his cases with an increase in the mortise. A wide joint is not stable and results in disturbed intra-articular pressure, which puts undue strain on the ligaments, capsule and cartilage.

The most frequent etiologic factor, both in our cases and in those reported in the literature, is an irregularity in the weight-bearing cartilage. This usually follows a marginal fracture, but may result from the astragalus being displaced laterally or mesially. Cartilage will not fill in these defects and produce a smooth surface.[7,8,9] The irregular surface destroys the smooth adjacent surface and produces a secondary arthritis.

The treatment of traumatic arthritis is primarily prevention. Fundamental preventive treatment is given at the time of reduction of the fracture. The reduction must be as nearly anatomic as possible. *X*-ray must show perfect reduction and restoration of joint width, especially in marginal fractures which are more often improperly reduced than are malleolar fractures. The fragment is usually displaced upward on the tibia and must be replaced to restore the continuity of the weight-bearing surface. If it is not replaced, traumatic arthritis results and fusion of the joint is called for to relieve the severe pain. Repeated attempts at closed reduction may be necessary; if they are not successful open reduction should be resorted to.

The next step in the prevention of traumatic arthritis is correcting malunited fractures before the articular cartilages have been severely damaged. The requirements in such reconstruction operations are: (1) restoration of the weight-bearing alignment; (2) restoration of the normal anatomic relationship between the articular surfaces of the tibia, fibula, and astragalus;

and (3) restoration of a satisfactory range of painless motion. If these requirements cannot be fulfilled, a fused ankle in the proper position is much more satisfactory. Speed[10] and Mackinnon reported poor results in attempting to reconstruct the articular surface of the tibia. They feel fusion is necessary to give a painless weight-bearing member. Wagner[11] reports uniformly poor results in reconstruction of the joint surface. A later arthrodesis was necessary in all seven of his cases.

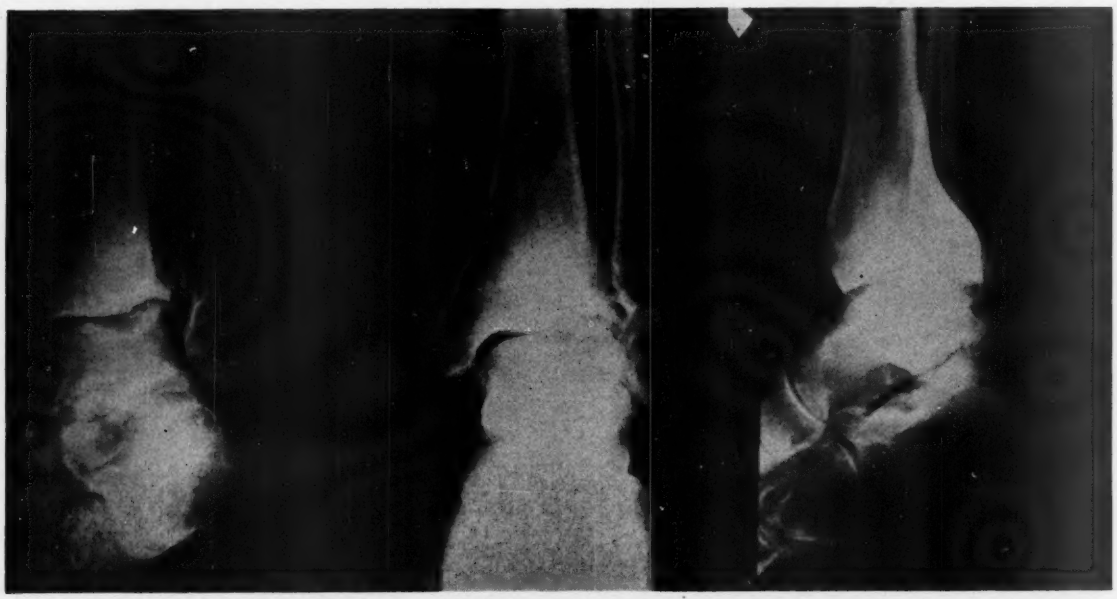

Fig. 5. I. H. Case v. Unsatisfactory reductions of three malleolar fractures producing osteoarthritis.

Fig. 6. A. E. Case vii. Unsatisfactory reductions of three malleolar fractures producing osteoarthritis.

The present study is based on eighteen patients who had secondary osteoarthritis following ankle fractures and desired relief of persistent pain and disability. We made an unsuccessful attempt to follow up and study, as a control series, a group of

patients with fractured ankles who had not returned with complaints. We were able to induce only two such patients to return.

3. *Symptom-Free Interval.* A perhaps significant similarity in this group is that sixteen of the eighteen had no symptom-

Fig. 7. E. E. Case VIII. Unsatisfactory reductions of three malleolar fractures producing osteoarthritis.

However, certain similarities in the eighteen post-fracture osteoarthritic patients provoke interesting lines of thought.

1. *Age.* Fourteen patients were between the ages of 35 and 56—that is, they were well within the age period of osteoarthritis. In the remaining four, there were special circumstances which seemed to afford adequate explanation of the onset at earlier ages: in two there were splitting fractures of the lower extremity of the tibia, where there were uneven articular surfaces; a third had a badly comminuted fracture in which three unsuccessful attempts had been made to obtain satisfactory reduction; the fourth patient was a waitress with an ununited mesial malleolus. The average age of the entire group of eighteen was 40.

2. *Sex and Weight.* Two-thirds of these patients (twelve) were women. Of these, five were recorded as overweight, and others may well have been so without mention of the fact in the history. The significance of this sex difference in incidence may lie in less strong muscles to support an injured ankle, and perhaps aggravation of this circumstance by overweight.

free interval between the time of fracture and the time they applied for relief of osteoarthritis.

4. Two-thirds of the patients had three malleolar fractures (two malleoli plus marginal), three had splitting fractures of the lower extremity of the tibia, and only two had two malleolar fractures.

5. Judging from the position at the time of their visit to us, only three of the eighteen reductions might be classified as nearly mechanically perfect. In three others there had been splitting fractures of the tibia, which left defects and irregularities in the lower tibial articular surface. The other twelve patients showed malalignments and deformities of various degrees.

6. Fourteen patients had no reduction or one or more closed reductions; three had closed reduction followed by open; the treatment received by the remaining patient is not known to us.

7. The condition at the time of their visit to us was such as to warrant fusion operations in nine out of the eighteen, and to place fusion under consideration in six others. Of the remaining three, no decision has been reached in one; one received

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NAME NUMBER	SEX	PRESENT AGE	WEIGHT	TIME OF INJURY	TYPE OF INJURY	TWO MALLEOLAR THREE MALLEOLAR SPLITTING	REDUCTION CLOSED OPEN
E.C. CASE 1	FEMALE	41	NOT RECORDED	10½ YEARS AGO	NOT KNOWN	THREE MALLEOLAR	CLOSED
E.OC. CASE 2	FEMALE	43	OBESE	1 YEAR, 8 MONTHS AGO	FELL ON ICE	THREE MALLEOLAR	CLOSED
H.P. CASE 3	FEMALE	36	OVERWEIGHT	3 YEARS AGO	SLIPPED ON RUG, FELL	TWO MALLEOLAR	CLOSED
S. F. CASE 4	FEMALE	35	LARGE, WELL DEVELOPED	7 MONTHS AGO	FELL ON ICE	THREE MALLEOLAR	CLOSED
I. H. CASE 5	FEMALE	50	MODERATELY OBESE	2½ YEARS AGO	STEPPING FROM AUTO	THREE MALLEOLAR	CLOSED
W. C. CASE6	MALE	36	NOT RECORDED	14 MONTHS AGO	FELL FROM TREE	SPLITTING OF TIBIA	CLOSED
A.E. CASE 7	FEMALE	44	NOT RECORDED	5 YEARS AGO	NOT RECORDED	THREE MALLEOLAR	CLOSED
E.E. CASE 8	MALE	43	NOT RECORDED	6 MONTHS AGO	FELL AND TWISTED ANKLE	THREE MALLEOLAR	IN BED 3 WEEKS NO MENTION OF REDUCTION
F. M CASE 9	FEMALE	48	SLIGHTLY OVERWEIGHT	3 YEARS AGO	NOT KNOWN	THREE MALLEOLAR	FIRST CLOSED THEN OPEN
M.L. CASE 10	FEMALE	42	NOT RECORDED	4 YEARS AGO	NOT KNOWN	THREE MALLEOLAR	NOT KNOWN
L.P. CASE 11	FEMALE	56	NOT RECORDED	2 YEARS AGO	FALL IN HOME	THREE-MALLEOLAR	CLOSED
H.R. CASE 12	FEMALE	21	NOT RECORDED	3½ YEARS AGO	NOT KNOWN	TWO MALLEOLAR	CLOSED
S.S CASE 13	MALE	31	NOT RECORDED	15 YEARS AGO	FELL TWO STORIES LANDING ON FEET	SPLITTING OF LOWER TIBIA	NOT REDUCED
R.S CASE 14	FEMALE	50	NOT RECORDED	15 MONTHS AGO	SLIPPED ON ICE	TWO MALLEOLAR	CLOSED OPEN 4 MOS LATER
J.S. CASE 15	MALE	22	NOT OVERWEIGHT	3 YEARS AGO	MOTOR ACCIDENT	TWO MALLEOLAR, AND SPLITTING OF LOWER TIBIA	THREE CLOSED REDUCTIONS
A.A CASE 16	MALE	46	NOT OVERWEIGHT	11 MONTHS AGO	FELL ON ICE	THREE MALLEOLAR	CLOSED
J.K. CASE 17	FEMALE	47	NOT RECORDED	17 MONTHS AGO	SLIPPED	THREE MALLEOLAR	CLOSED
A.V.P CASE 18	MALE	28	NOT RECORDED	13 MONTHS AGO	FELL TWO STORIES TO PAVEMENT	THREE MALLEOLAR	DEBRIDEMENT, THEN CLOSED AFTER 10 DAYS. OPEN 4 MO LATER, SCREW IN INTERNAL MALLEOLUS

FOLLO

L.C. CASE 1	FEMALE	51	OBESE	8 YEARS AGO	FELL DOWN STAIRS	THREE MALLEOLAR	CLOSED
J.A.F. CASE 2	MALE	68	NOT RECORDED	4½ YEARS AGO	SLIPPED ON ICE, TWISTED ANKLE	THREE MALLEOLAR	CLOSED

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THRITIS CASES

ORIGINAL DEFORMITY ORIGINAL REDUCTION	INTERVAL FREE FROM SYMPTOMS	PRESENT X-RAY FINDINGS	TREATMENT INSTITUTED OR ADVISED
X-RAYS NOT SEEN	UNTIL 3 MONTHS AGO	GOOD REDUCTION EXCEPT FOR SLIGHT VARUS DEFORMITY. EVIDENCE OF FAIRLY EARLY OSTEOARTHRITIS	TO CORRECT VARUS. IF SYMPTOMS NOT RELIEVED, TO CONSIDER FUSION.
X-RAYS NOT SEEN	NONE. SWELLING AND PAIN WORSE RECENTLY	BAD REDUCTION. SPREADING OF MORTISE. BACKWARD DISPLACEMENT OF FOOT. NON-UNION OF MESIAL MALLEOLUS. MARKED OSTEOARTHRITIS	FUSION PERFORMED
X-RAYS NOT SEEN	NONE	BAD REDUCTION. BACKWARD AND UPWARD DISPLACEMENT, VALGUS. MARKED OSTEOARTHRITIS	FUSION PERFORMED
X-RAYS NOT SEEN	NONE	MODERATELY BAD REDUCTION. LATERAL DISPLACEMENT AND VALGUS. DEFINITE OSTEOARTHRITIS	NOT KNOWN
X-RAYS NOT SEEN	NONE, WORSE PAST FEW MONTHS	MODERATELY BAD REDUCTION. FOOT LATERAL AND POSTERIOR, VALGUS. SEVERE OSTEOARTHRITIS	FUSION PERFORMED
X-RAYS NOT SEEN	NONE	VERTICAL SPLITTING FRACTURES, SAGITTAL AND CORONAL PLANES, ANTERO-POSTERIOR AND LATERAL SPREADING. MODERATE OSTEOARTHRITIS	FUSION PERFORMED
X-RAYS NOT SEEN	NONE	BAD REDUCTION. LATERAL DISPLACEMENT AND VALGUS MODERATE OSTEOARTHRITIS	FUSION PERFORMED
X-RAYS NOT SEEN	NONE	BAD LATERAL DISPLACEMENT AND VALGUS SLIGHT OSTEOARTHRITIS	TO TRY PHYSIO-THERAPY FOR 6 WEEKS. IF NOT RELIEVED, FUSION TO BE ADVISED
X-RAYS NOT SEEN	NONE	1. BEFORE OPEN REDUCTION. SLIGHT LATERAL DISPLACEMENT AND VALGUS. NON-UNION OF MESIAL MALLEO.. 2. AFTER OPEN REDUCTION: UNION, SLIGHT LATERAL DISPLACEMENT & VALGUS. PROBABLY EARLY OSTEO-ARTH.	STILL UNDER OBSERVATION
X-RAYS NOT SEEN	PROBABLY NONE	GOOD REDUCTION. GOOD ALIGNMENT POSSIBLY SLIGHT MORTISE SPREAD DEFINITE OSTEOARTHRITIS	TO TRY CONSERVATIVE MEASURES
X-RAYS NOT SEEN	NONE	GOOD REDUCTION. GOOD ALIGNMENT THIN CARTILAGES ENORMOUS SWELLING DEFINITE OSTEOARTHRITIS	CONSERVATIVE TREATMENT TO BE TRIED ADVISED THAT ONLY REAL RELIEF WILL BE BY FUSION.
X-RAYS NOT SEEN	NONE	NEARLY GOOD REDUCTION. NON-UNION OF INTERNAL MALLEOLUS. SLIGHT OSTEOARTHRITIS IN ASTRAGALAR-INTERNAL MALLEOLAR ARTICULATION. SWELLING.	FUSION ADVISED BECAUSE OF FINDINGS AND BECAUSE OF OCCUPATION OF WAITRESS
NO ORIGINAL X-RAYS	3 YEARS SYMPTOMS 8 YEARS FREE 4 YEARS SYMPTOMS	WIDENING OF LOWER EXTREMITY OF TIBIA. DEFORMITY AND ROUGHENING OF ARTICULAR SURFACE. MODERATE OSTEOARTHRITIS	TO TRY CONSERVATIVE TREATMENT. WILL PROBABLY NEED FUSION
ORIGINAL DEFORMITY GREAT CLOSED REDUCTION BAD. OPEN REDUCTION IMPERFECT	NONE	MODERATE BACKWARD DISPLACEMENT OF FOOT. SLIGHT VALGUS. CARTILAGES GONE. MODERATE SCLEROSIS	FUSION ADVISED
VERY SEVERE ORIGINAL DEFORMITY	NONE	MUCH JOINT DEFORMITY. VERY BAD ALIGNMENT. ROUGH AND OBLIQUELY PLACED ARTICULAR SURFACES	FUSED
SEVERE ORIGINAL DEFORMITY	NONE	BAD REDUCTION. MARKED LATERAL DISPLACEMENT OF LOWER FRAGMENTS AND FOOT. MARKED VALGUS. THIN CARTILAGES.	FUSED
SEVERE ORIGINAL DEFORMITY	NONE	BAD REDUCTION. LATERAL DISPLACEMENT OF LOWER FRAGMENTS AND FOOT. BACKWARD DISPLACEMENT OF FOOT DEFINITE OSTEOARTHRITIS	FUSED
X-RAYS NOT SEEN	NONE	MARKED VARUS DEFORMITY. SCREW IN INTERNAL MALLEOLUS, PROBABLY UNION. MUCH OSTEOARTHRITIS	FUSED

W-UP CASES

SEVERE ORIGINAL DEFORMITY. EXCELLENT REDUCTION		GOOD UNION. GOOD ALIGNMENT. SLIGHT THINNING OF CARTILAGES LATERALLY	COMMENT: GOOD RESULT. SOME PAIN IN ANKLE WITH CHANGE IN WEATHER. SOME PAIN AND SWELLING WITH PROLONGED USAGE
VERY SEVERE DEFORMITY. EXCELLENT REDUCTION.	PAST 3 YEARS	GOOD UNION. GOOD ALIGNMENT NO OSTEOARTHRITIS	COMMENT: EXCELLENT RESULT. NO SYMPTOMS. PAST THREE YEARS AS GOOD AS OTHER ANKLE

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conservative treatment; the treatment of the third is not known.

8. Thirteen of the patients applied for relief of disability from six months to three years following fracture. Analysis of the remaining five (who came in three and one-half to fifteen years after fracture) on the basis of age at the time of injury and perfection of reduction, is interesting. (Table 1.)

TABLE 1

		Age at Injury	Interval since Fracture, Years	Reduction	Possible Explanation of Interval
H. R.	Case XII	18	3½	Bad; non-union of internal malleolus	Youth at time of injury
M. L.	Case X	38	4	Good	Good reduction
A. E.	Case VII	39	5	Bad	None
E. C.	Case I	30	10½	Good	Good reduction, comparative youth
S. S.	Case XIII	16	15	Bad splitting fracture	Youth at time of injury

Only one of these cases with interval longer than three years is not susceptible of explanation on either the grounds of unusual youth or unusually good reduction.

In further consideration of age as a factor, it may be noted that two of the three cases we classified as having a nearly mechanically perfect reduction were in the above group, M. L. and E. C. The age of the third, L. P., Case XI, who applied to the hospital for relief two years after her fracture, is a likely explanation for her non-appearance in the more delayed group. She was the oldest patient in this series, and was 54 at the time of her fracture.

SUMMARY AND CONCLUSIONS

Although conclusions are unwarranted in such a small series of cases, several interesting possibilities are suggested, which time and further experience may establish as facts.

1. Age, sex, and probably weight appear to be definite factors in the occurrence of these fractures and in determination of their outcome.

2. Protracted symptoms over a period of months following treatment constitute a very unfavorable sign.

3. Three malleolar fractures and splitting fractures of the lower extremity of the tibia seem more prone to lead to secondary osteo-arthritis than fractures of one or two malleoli.

4. Nothing short of mechanically perfect reduction affords a reasonable chance of avoiding secondary osteoarthritis, and even then there is no positive assurance of escape.

5. Perfect reduction will sometimes bring excellent results, even when all other factors are unfavorable, as shown by our two follow-up cases. Between them, these two cases embodied all the unfavorable features: age (63 and 43), sex, obesity, three malleolar fractures with severe original deformities, symptoms persisting for one and a half years after injury. They had perfect closed reductions, and showed excellent end results four and a half and eight years after injury.

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ACTIVE MUSCLE USAGE IN THE TREATMENT OF FRACTURES

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IN the treatment of fractures proper reduction of the lesion, immobilization until union occurs, and restoration of the function of the extremity are recognized necessities. It is also generally accepted that the optimum time for reduction of a fracture is as soon as possible after the accident. At this time contractures are not present, the tissues are not indurated, and swelling is not extensive. The reduction is accomplished by the use of manipulation, traction, gadgets, or open operation. The permanent immobilization is immediately applied whether it be plaster of Paris in any form, or skeletal traction in splints or plaster of Paris.

The object of this paper is to present some facts not given sufficient attention in the treatment of fractures. We were taught in school to immobilize a fracture, and to keep the muscles quiet. This treatment quickly leads to stiff joints, swelling of the extremity and a convalescence prolonged long after the bony injury has healed. This teaching is fallacious. During the entire time of immobilization of the fracture, active motion of all joints not immobilized and muscle contraction of those beneath the fixation dressing prevent joint stiffness and swelling.

To secure active joint motion of unimmobilized joints the fracture must be adequately fixed so as not to cause pain. If this is not done, any attempt to move the joints causes pain and muscle spasm, and no motion occurs. Active contraction of all muscles under the dressings causes active relaxation in their antagonists, the tonicity of these muscles is maintained, and the minimum amount of atrophy occurs. Active usage is of prime importance during the fixation period of healing. This principle is not sufficiently stressed.

How many times have you seen a frozen shoulder following a Colles' fracture, or after some trivial accident to the elbow?

How many times have you seen stiff fingers and a swollen hand following a Colles' fracture?

How many times have you seen extreme atrophy of the quadriceps following a fracture of the patella?

How many times have you seen contracted toes and an acute anterior metatarsalgia following fracture of the metatarsals?

The cause of each of these complications is lack of active muscle usage. These disabling sequelae can be prevented.

Every patient should actively move joints which are not immobilized and contract the muscles under the dressings from ten to twenty times or for ten minutes every hour on the hour during the entire waking day beginning the day of the injury and continuing until the fracture is healed. This simple rule will prevent these stiff joints and swollen extremities that take months to rehabilitate.

In injuries to the phalanges and metacarpals adequate fixation of the involved fingers only is needed and active use of all others is permitted.

In Colles' fractures, fractures of both bones of the forearm, and other fractures of the radius or ulna, the immobilization extends from just proximal to the distal palmar crease to the axilla. Complete active motion of all the fingers and shoulder is done hourly, together with biceps contraction. This active use keeps down swelling and keeps the muscle-tendon sense normal. Joint stiffness then will not occur

in the fixed joints. The fracture must be kept adequately immobilized to prevent pain, for this prevents active usage.

The same activity is insisted upon even when a shoulder spica has been applied.

In the lower extremities, active toe motion and quadriceps exercises hourly will keep the muscle-tendon sense normal, prevent atrophy of the muscles, and prevent joint stiffness.

No passive motion or any other form of physiotherapy will then be needed. After complete healing of the fracture and removal of all fixation, joint function returns rapidly and completely with a few hot soaks in the bathtub.

Immediately on removal of the fixation apparatus after this régime, active motion in the immobilized joints through 30 to 40 degrees of painless motion is possible.

SUMMARY

This paper stresses the importance of active muscle and joint usage in the treatment of fractures neglecting other forms of physiotherapy.

OCCUPATIONAL therapy is almost essential in the care of the convalescent orthopedic patient. These patients are often in good general health, vigorous, alert, and restless, and their physical disability, which may be confined to a portion of one extremity, is likely to become irksome.
From—"Convalescent Care" (New York Academy of Medicine).

TUMORS RELATED TO CARTILAGINOUS GROWTH

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IT is the growing opinion of many that neoplasia is a mechanism transmitted as a recessive character by heredity, fixed in the tissues to come in bloom at a certain period of the individual's development if the individual survives. The group of bone tumors reported here serve as excellent illustrations of this theory. It is believed that any other group of malignancies might exhibit similar truisms if classified according to their embryonic anlagen.

Taxonomy of Bone Tumors:

I. Tumors related to cartilaginous growth:
   - Bone cysts
   - Giant cell tumor
   - Osteolytic sarcoma.

II. Tumors related to precartilaginous growth:
   - Osteogenic sarcoma
   - Chondroma
   - Osteochondroma.

III. Tumors not primarily of osseous origin:
   - Ewing's tumor
   - Multiple myeloma
   - Metastatic carcinoma
   - Fibrosarcoma.

This classification[1] and most of the ideas presented in this paper are based on the work of Bloodgood, Geschickter, Copeland,[2] and Nélaton.[3]

Bone Cyst. The clinical features of bone cyst as manifested in the fourteen patients who received this diagnosis at the State University of Iowa are as follows:

Average age	13.6 years
Females	9
Males	5
Duration of symptoms	2.9 years
Incidence of trauma	7
Incidence of fracture	4
Pain	11
Tenderness	11
Swelling	11
Redness	3
Multiple lesions	5

No lesion was found in a bone that was not derived from cartilage. The characteristic features of the roentgenograms were: ununited epiphysis, metaphyseal location, central area of bone destruction crossed by trabeculation. If there had been a fracture, a dense shadow of new bone was seen. The shell was not perforated except by fracture or surgery and its thickness varied inversely with the duration of symptoms. There was a fibrous tissue lining and the contents might be fluid, fibrous, grumous material or an admixture of all. Fibrous trabeculation could be felt; there was multiloculation and occasionally bone spicules. Microscopically, giant cell areas were seen, areas of old hemorrhage, bone islands, and areas of fresh hemorrhage.

When a bone cyst occurs at a site where multiple centers of ossification are present, it may take on many of the characteristics of a giant cell tumor and tend to progress. Such a lesion will present multiple small cysts filled with hemorrhage and surrounded by giant cells. However, it is metaphyseal in location. Such a patient (G. B.) at age 22 complained of pain, swelling, and tenderness in the upper femur of eighteen years' duration. She responded poorly to bone graft, immobilization, and Roentgen therapy.

The line of demarcation between the single bone cyst, the giant cell variant of bone cyst, and polycystic osteititis fibrosa is by no means sharp. The histology is similar and patients may be found who are difficult to classify. In one such case (L. T.), the patient, aged 29, first complained of pain in the right hip. She was found to

have two small bone cysts in the body of the right ilium which became asymptomatic for ten years following Roentgen therapy. She was then found to have bone cysts of the fourth, fifth, sixth and seventh right ribs and a mild pleurisy. Biopsy revealed osteititis fibrosa cystica, the symptoms of which were relieved following Roentgen therapy. The blood calcium was normal.

The production of a bone cyst seems to be bound up with the activity of the giant cell. These cells may run amok when the reparative power of bone is jeopardized by a decalcifying process such as hyperparathyroidism, but when the agent that injures bone is trauma, the giant cells are allowed only a short period of activity before healing occurs and we get the simple cyst. Victims of hemophilia may bleed into bone and cause mimic osteitis fibrosa. Many cases of Paget's disease resemble the generalized type of bone cyst; the common factor is the giant cell.

Giant Cell Tumor. The clinical features of giant cell tumor as manifested in the twenty-two cases in which this diagnosis was made at the State University are as follows:

Average age	28 years
Females	11
Males	11
Duration of symptoms	16 months
Incidence of trauma	15
Incidence of fracture	13
Pain	20
Tenderness	21
Redness	4
Multiple lesions	1

The characteristic *x*-ray features were asymmetrical location in the epiphysis, osteolysis and expansion centrally at the expense of cancellous bone. Trabeculation was seen, but there was no periosteal reaction. The fibrous tissue lining resembled that of bone cysts and the contents were usually hemorrhagic, oozing blood, friable and fibrous trabeculations. Bone spicules were palpable. Microscopically there were large masses of giant cells in a mass of smaller round cells. The giant cells were more numerous about areas of old hemorrhage, spicules of bone and cyst walls. The nuclei of the round cells had the same morphology and staining characteristics as the nuclei of the giant cells. Spindle cells indicated a healing reaction. Some giant cell tumors are practically indistinguishable from osteititis fibrosa cystica because of this fact. The lesions occurred in bones where the anatomic structure was unusually prominent. As an example, we may cite the case of a patient who at 59 presented a giant cell tumor of the phalanx which had been present forty-four years as a sequel to a baseball injury.

Bloodgood, Geschickter and Copeland hold that these lesions can arise only from bone derived from cartilage. Two of the patients of this series had giant cell tumor of the skull, one of which was located at the angle of the mandible, a frequent site, and the other was said to be in the maxilla, a bone formed in membrane. However, a description of the lesion, as given in detail at the time of operation, located the tumor in the roof of the nose and the antrum. A similar problem had confronted the above authors, whose case involved the antrum and orbit. Their argument was that the ethmoid, which is derived from cartilage, might well be the source of the neoplasm since the lamina papyracea forms the medial wall of the orbit and the uncinate process overlies the maxillary ostium. Curiously enough we have seen two patients with osteitis fibrosa cystica of the skull and each had a lesion at the angle of the jaw and another lesion in the ethmoid, one presenting a visible swelling just to the right of the nose and another at the nasion. Nélaton presents beautifully hand drawn pictures of giant cell tumors, one of them limited to the ethmoid bone.

Giant cell tumor is occasionally found in the vertebrae and may then cause symptoms of cord compression. Lewis[4] has noted that such lesions are of the spindle cell variety and have a tendency to heal on their own, some patients having gone on to healing subsequent to curettage of a large

part of the tumor. A fusion operation may follow.

When a giant cell tumor is present

Fig. 1. Typical bone cyst. Ununited epiphysis, metaphyseal location, central area of bone destruction crossed by trabeculation.

directly beneath the periosteum, it may pulsate. Its rate of growth may be very rapid and cause perforation of the cortex. A mistaken diagnosis of osteogenic sarcoma is often made in such cases and amputation performed where more conservative therapy would have sufficed. This happened to one of the patients of this group who at age 35 developed a pulsating lesion over the left medial tibial condyle two months following a blow with a hammer. Biopsy was erroneously omitted and no inkling of the true diagnosis was available until the report was returned from the pathologist.

Giant cell tumors occur in elderly individuals but these are either of the spindle cell variety with a history of long duration or there is a remnant of the epiphysis. Roentgenograms of other than involved bones should be taken.[5]

Microscopy divides epulis into a fibrous and a giant cell variety homologous to osteititis fibrosa and giant cell tumor. It is said that the alveolar border is a veritable epiphyseal line, the deciduous teeth like cartilage, the odontoclasts comparable to osteoclasts, multinucleated, and serving the same purpose in absorbing the mem-

Fig. 2. Bone cyst following fracture. If there has been a fracture, a dense shadow of new bone formation is seen.

branous bone that serves as anchorage for the deciduous teeth. This accounts for the fact that epulis occurs in the young and never at the site of molars. In giant cell tumors of the tendon sheaths it is necessary to demonstrate some bony structure of cartilaginous origin. This Geschickter and Copeland have done. Giant cell tumors have been found to arise in the patella, a sesamoid bone. The giant cell xanthomata are tumors of sesamoid bones.

Malignant giant cell tumor is of two types: those that recur locally and those that metastasize. Although giant cell tumors do have a tendency to recur locally, this is usually due to incomplete removal, poor selection of treatment, or both. Metastatic tumors are usually mistaken diag-

noses—the lesion is an osteolytic sarcoma rather than a giant cell growth.

Osteolytic Sarcoma. There are two types of osteolytic sarcoma, both rare. The *chondroblastic* sarcoma arises from a proliferation of cartilage at the epiphyseal line during puberty. Roentgenographically there is a mottled area of bone destruction with or without a slightly expanded bone shell. There is a definite periosteal reaction. Microscopically these lesions show a proliferation of chondroblasts which produce abortive areas of calcifying cartilage. At the margin of the tumor giant cells proliferate as a defensive reaction and attempt to remove calcified products of the tumor. The usual duration of the disease from onset until death is fifteen months. The finding of cartilage rules out giant cell tumor.

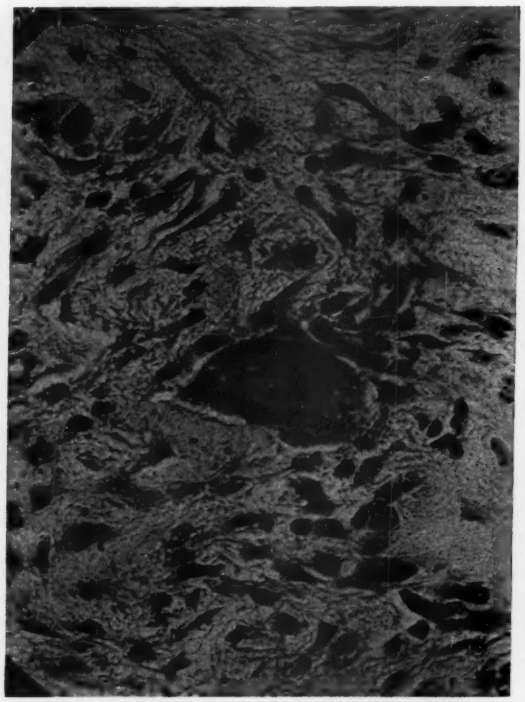

Fig. 3. The spindle cell variant of giant cell tumor. The spindle cells indicate a healing reaction. This patient had had the tumor forty-four years as a sequel to a baseball injury of the phalanx.

The *true* osteolytic sarcoma arises within the marrow in the shaft of long bones, shows a wide age distribution, produces pathologic fracture, and is frequently a pulsating lesion. Roentgenographically there is a central area of bone destruction without expansion of the cortex and without periosteal involvement. Microscopically, there are large spindle cells, rounded osteoblasts with numerous mitotic figures and a small amount of poorly formed osteiod tissue. The giant cells present in the lesion represent an attempt to remove the incompletely formed osteiod tissue. The average duration from the time of onset until death is twenty-two months. There are no cases of osteolytic sarcoma on record at the State University of Iowa Hospitals.

Fig. 4. Osteititis fibrosa cystica of the chondrocranium. It is believed that the lesion to the right of the nose must have arisen from the ethmoid bone.

The rôle of trauma in the production of giant cell tumor is not always obvious, particularly when the lesion is in the chondrocranium. In the more rapidly growing types, such as the subperiosteal giant cell tumor, trauma plays a more obvious part. Certainly these lesions are related to the metabolism of the epiphysis and one finds

it hard to believe that the giant cell is not the essential part of the lesion. Given factors that will produce an environment favorable to the existence of the giant cell and unfavorable to the processes of repair, one may expect the formation of a giant cell tumor. Hyperparathyroidemia producing a calcium deficiency of bone will jeopardize the process of repair and permit the osteoclast to proliferate. Trauma may have a similar but less marked effect. Bleeding into bone where the only factor present to stop the bleeding is a pressure equivalent to the systolic blood pressure may, in a hemophiliac, give a similar picture.

The tendency for a metabolic imbalance to promote neoplasia is well known in medicine and has been emphasized by Trout and Kunath.[6] Fifteen patients, reported by the former author all had carcinoma of the breast, were treated with radical mammectomy, subsequently became pregnant and immediately developed carcinoma of the remaining breast, all dying a rapid and fulminating death.

Broders[7] raises the question as to whether malignancies are ever localized diseases. Carcinoma of the breast is related to the pregnancy hormones; tumors related to cartilaginous growth are stimulated by the parathyroid hormones. Everyone is aware of the work of Slye on the hereditary aspects of cancer. Broders, after a study of cancer in identical twins, has reached the conclusion that the stage for malignancy is prepared beforehand and, if the patient is alive when the time for cancer arises, he will suffer from it. He illustrates with one very interesting set of twins 92 years of age both of whom developed identical lesions in the left breast of identical grade of malignancy.

Geschickter voices this same idea in discussing tumors related to cartilaginous growth. "From a clinical standpoint the relationship between normal development and tumor formation makes the lesion and the age at which it occurs a most important consideration in the diagnosis and treatment of the tumor. In fact, with few exceptions, the nature of the neoplasm with which the clinician is dealing can be

Fig. 5. Subperiosteal giant cell tumor of the tibia. This was a pulsating lesion, the cortex was perforated, but there was no periosteal reaction. An erroneous diagnosis of osteogenic sarcoma was made and amputation performed. Local removal would have sufficed.

readily restricted to a limited number of possibilities of the nature of the tissue development at the age and site where the tumor occurs is given adequate consideration."

That cancer is inherited does not explain the mechanism of its production. It is believed that the endocrines play an even more subtle part in the production of neoplasia than is usually conceded.

In the treatment of these lesions little has been added since the time of Nélaton other than the use of radiation therapy. Nélaton stated in reference to therapy:

"31. Extirpation, pure and simple, merits almost no confidence. If extirpation

consists of simple excision of the tumor, or even of the breaking up, uprooting, or finger extraction, it almost never (unless perhaps in the encysted, enucleated variety) succeeds in securing completely disappearance of the lesion; extirpation constitutes most often, as long as there remains the least shred of tissue adherent to the bone substance, merely a palliative and illusory operation, soon followed by recurrence.

"32. The application of hot irons is equally futile, in most cases. . . .

"33. Scraping, crumbling with a gouge, and above all, chemical caustics, particularly zinc chloride (Conquoin paste) carry out the procedure more efficiently.

"34. Extirpation combined with scraping or with potential cauterization affords hope for true success, at least as far as subperiosteal tumors are concerned."

Interestingly enough, no patient described in this series recieved the combined treatment of curettage, chemical cautery, bone graft, immobilization and Roentgen therapy.

CONCLUSIONS

A survey of tumors related to cartilaginous growth is presented with emphasis on the similarity of these lesions and the inclusion of the generalized type of osteitis fibrosa cystica in the classification, thus relating these tumors to the endocrine mechanism. Stress is placed on the fact that these neoplasm occur at certain ages, the activity of the osteoclast and its ability to become malignant or benign depending on its stimulant.

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THE COMPLICATIONS OF EMPYEMA IN CHILDREN*

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THE Empyema Commission of the United States Army promulgated certain principles in the treatment of empyema thoracis which have greatly lowered the mortality of that disease. We believe, with Bohrer, Heuer, Graham, Berck and others, that the mortality of uncomplicated empyema should be practically nil if these principles of adequate drainage at the proper time are followed, and that it makes no difference what manner of surgical attack is used as long as it follows these principles. The complications are, therefore, responsible for the death rate and for a prolonged convalescence. Thus, in the present state of our knowledge, the complications are of greater importance than the disease.

We have made a study of the complications arising in the cases of empyema occurring on the Children's Surgical Service at Bellevue Hospital, New York City, during the past ten years. Bohrer has already reported on some of these cases in a paper dealing with the general aspects of empyema in children; the present communication will therefore deal entirely with the complications. There are 248 cases in the series, in 179 of which there were 283 complications, thus leaving only sixty-nine cases—slightly more than one-quarter—without complications. Most of the patients received their entire treatment at Bellevue, the others arriving at Bellevue after having been in one or more hospitals during the previous stages of their disease. This may make our figures for complications a little above the average but it also gives us more cases for discussion.

Since empyema is itself a complication of some other disease it may be difficult to tell whether the complication under discussion is that of the empyema or of the primary disease. For the purposes of this paper those conditions are considered as complications which arose after the empyema had become established or those existing before the onset of the empyema but which had an effect on its course.

TABLE I

PRINCIPAL CAUSE OF DEATH

Septicemia	9
Exhaustion	4
Pneumonia	4
Brain abscess	2
Peritonitis	2
Tension pneumothorax	2
Meningitis	1
Pulmonary tuberculosis	1
Ruptured trachea	1
Shock	2

Table I gives the principal cause of death in the twenty-eight fatalities. It will be noted that empyema per se is not listed and the only death that in any way could be considered as due to empyema was that of shock. This occurred in a six weeks' old infant on whom it became necessary to do a simple intercostal closed drainage to relieve dyspnea caused by a great collection of pus in his chest. Three other patients died before the empyema had reached the operable stage, which reduces the mortality in surgical empyema to twenty-five cases.

Table II gives the occurrence of the principal complications and the number of times each occurred among the fatalities, regardless of whether it was considered the chief cause of death or only a secondary factor. This will explain the discrepancy between the two tables.

Septicemia is seen to be the most serious complication and the one responsible for the greatest number of deaths. In the fatal

* From the Children's Surgical Service, Bellevue Hospital, New York City, Fenwick Beekman, M.D., Surgeon-in-Charge.

cases the blood culture showed hemolytic streptococcus in each instance. In six cases, which include the three nonoperative cases mentioned above, the chest fluid also contained the same organism. In the other three, culture of pus from the chest showed pneumococcus. Two patients with positive blood culture recovered. One had a *Streptococcus viridans* and the other, hemolytic streptococcus. Several patients had a positive pneumococcus blood culture during the antecedent pneumonia, but the blood culture was sterile before the onset of the empyema.

TABLE II
COMPLICATIONS

Complication	Number	Deaths*
Septicemia	11	9
Peritonitis	4	4
Pericarditis	2	2
Meningitis	3	3
Brain abscess	2	2
Brain embolus	1	0
Exhaustion	4	4
Secondary infection	6	3
Contagious diseases of childhood	26	1
Otitis media and mastoiditis	35	4
Pneumonia	14	5
Bilateral empyema	7	0
Bronchiectasis	7	0
Lung abscess	3	1
Inadequate drainage	59	3
Osteomyelitis of rib	27	0
Tuberculosis	6	1
Bronchopleural fistula	31	7
Chronic empyema	12	0
Late recurrence	4	0
Cellulitis of chest wall	4	1
Perforation of diaphragm	3	0
Miscellaneous	12	3

* The figures in this column do not correspond with those in Table 1, because every complication associated with a fatality is listed, not just that primarily responsible for each death.

Pericarditis and peritonitis as complications are simply local manifestations of the general septicemia. Both patients with pericarditis had positive blood cultures. The two with peritonitis also had a positive blood culture, and in the other two blood culture was not taken.

Meningitis would naturally be considered as a result of a septicemia and in two of the three cases positive blood cultures were obtained. In the third no organism could be grown from the blood. All were fatal.

The treatment of these conditions does not differ from their treatment under other circumstances. Since the hemolytic streptococcus plays such an important rôle, sulfanilamide should be tried, but in the presence of an overwhelming infection too much should not be expected of it. Blood transfusions may be of value.

Brain abscess frequently has its genesis in some intrathoracic suppuration, but the frequency of empyema as a cause varies with different investigators. Thus Nickerson found it in only one case out of twenty, Schorstein in fifteen out of sixty-nine, and Cohen in four out of nineteen. In our series brain abscess occurred twice and each time was fatal, although in one case it was possible to localize and drain the abscess. This prolonged life but did not alter the ultimate outcome.

One child apparently had two attacks of cerebral embolus but recovered. This was a 7 year old boy who developed postoperative pneumonia after appendectomy for acute appendicitis. He apparently recovered but returned to the hospital two weeks after discharge suffering from pleurisy with effusion. This progressed to empyema which was drained three weeks later by rib resection. On the twenty-fifth day after the empyema operation he suddenly developed headache, convulsions, dyspnea, cyanosis, nystagmus and pathologic reflexes, and went into coma. In about a day he began to improve and in three days seemed completely recovered. Nineteen days later he repeated the same performance and likewise recovered. The neurologists made the diagnosis of cerebral embolus.

The patients with brain abscess followed the same chain of events, but finally died. Streptococcus viridans was found in the empyema pus of each of these three cases

and also in the pus from the brain abscess which was drained.

In a disease which is always a complication, and in most instances may be but one of several complications, exhaustion must play an important part. Maes and his associates found that 40 per cent of the deaths were due to exhaustion and toxemia. In our series exhaustion seemed imminent in most of the cases and in four appeared to be the deciding factor in a fatal outcome. Children withstand a prolonged infection poorly and the younger the child the lower the resistance. By the time a child has passed through the various illnesses which precede operable empyema he is pale, weak, listless and irritable. He has a certain amount of fever and is obviously toxic. A case in point is that of a six months old boy who had in turn varicella, retropharyngeal abscess, cervical lymphadenitis, lobar pneumonia, bilateral otitis media and finally empyema. The empyema was drained by means of a flapper tube in an intercostal incision, but the child developed a pyocyaneous infection in his wound in addition to the original staphylococcus infection and died.

To combat this exhaustion we build up the fluid reserve which is nearly always depleted, transfuse the patient, and put him under the care of special nurses. The simplest possible operation is done, but even then some of these patients die.

In any children's service the contagious diseases of childhood are dreaded complications—dreaded alike for their effect on the patient and on the other inmates of the ward. These diseases appear as complications in twenty-six instances in this series. Measles was the most frequent, with sixteen cases, among which was one fatality. The other diseases were found in only one or two instances each. Measles developing before the empyema is not considered a complication although there were eight such cases in this series. It is obvious that the relationship between empyema and a complicating contagious disease of childhood is only casual.

On the other hand acute otitis media can be considered to have a direct relationship to the empyema or its antecedent infection. As would be expected this was a common complication occurring thirty-five times. Of these, six developed mastoiditis and four died. A great many other patients had running ears which started before the empyema and are not listed as complications. It is hardly necessary to state that ears should be inspected first in trying to determine the cause of an unexpected temperature rise.

Pneumonia as a complication of empyema may be an extension or recurrence of the original pneumonia; or it may be a new disease on the opposite side. It is a particularly serious complication with a 36 per cent mortality in our cases. Naturally, a severe disease such as pneumonia, arising in a child already approaching exhaustion from a long battle with infection brings with it a high death rate. The second pneumonia may occur before the empyema is ready to be drained or it may appear late when the empyema is nearly well. As would be expected, the later in the postoperative course that the second pneumonia appears the greater the chances for recovery.

When empyema follows the second pneumonia thus making a second empyema the prognosis is good. Apparently a patient who has sufficient resistance to combat the second pneumonia long enough to form empyema will recover under proper treatment. Thus, of fourteen patients with secondary pneumonia five died and seven developed empyema. There was no fatality among the seven cases of double empyema although in one instance the second empyema followed the first so closely that they were drained on successive days.

Bronchiectasis may be cause by repeated attacks of pneumonia and it is not unlikely that during some one of these attacks of pneumonia empyema will develop. Likewise, lung abscess may be a cause of bronchiectasis and may also cause empyema by perforation into the pleural cavity.

Seven of our patients had empyema as a result of one of these conditions which in turn was followed by bronchiectasis. There was no connection between the empyema and the bronchiectasis except that they were both complications of another disease.

We now come to a series of complications directly related to the operation rather than the infection. Insufficient drainage or pocketing of sufficient importance to require a secondary operation occurred fifty-nine times—the most common complication of the entire series. Three patients died, but other complications were responsible for the fatality and the pocketing was of minor significance.

Bohrer has already described our method of surgical attack and I will not repeat it other than to say that while we limit ourselves to no particular types we usually do an intercostal thoracotomy or rib resection using a flapper tube as a drain. As most of the cases requiring secondary operation had simple intercostal drainage in the first instance it might be thought that a more radical operation such as rib resection would be a better procedure. This we do not believe to be the case.

By the time a child has developed empyema in an operable stage he has been through a long, serious illness and is in a weak and toxic condition. The undrained infection causes toxemia much greater than the temperature would indicate and the patient is in no condition to tolerate anything but the quickest and least traumatizing operation.

Intercostal drainage can be done under local anesthesia, in bed, with a minimum of shock. It is therefore ideal for these patients and is often a life-saving procedure. Even if the drainage does prove to be insufficient it has allowed the patient to improve enough so that later he can stand the more extensive procedure of rib resection with safety.

Blocked drainage tube from fibrin plugs or rising diaphragm may allow the granulations to wall off a cavity at a distance from the tube. Too early removal of the tube can cause the same thing. In this connection it must be remembered that the pleural cavity may be able to tolerate a large amount of pus without giving a febrile reaction and hence the responsible surgeon should inspect the dressings frequently to be sure that the drainage is adequate. If he waits for a temperature rise it may be too late. Drainage tubes should not be removed until the cavity has been obliterated, for the same reason.

Occasionally the empyema is loculated and the intercostal drainage will drain only one pocket. Then the fibrin plaques will have to be left until they liquefy. Should they organize before this happens, secondary operation becomes necessary. That this is not a frequent condition can be seen by the fact that fully half of the cases with uncomplicated recovery had only intercostal drainage.

Late recurrence is not particularly uncommon and appeared four times in this series. No fatalities occurred and the condition responded promptly to adequate drainage. The original empyema had drained longer than usual in each case so it is probable that a more complete drainage in the first instance would have prevented the late occurrence. Six years was the longest interval in our series but Meade reports a case recurring after ten years.

Osteomyelitis of the rib is listed as a complication in twenty-seven cases of which sixteen had intercostal drainage. Pressure of the tube against the adjacent ribs may well have been a cause in the intercostal drainage cases. In many other instances the operator described a pocket adjacent to the osteomyelitis in a sinus of long standing and the impression is inescapable that the undrained pocket was responsible for the failure to heal rather than the bare bone felt by the examining probe. Without the underlying pocket the osteomyelitis would probably have taken care of itself. This does not apply to frank osteomyelitis with sequestration which is not particularly rare although probably not so

frequent as our figures would seem to indicate.

With respect to chronic sinuses, consideration of the six cases in which tuberculosis appeared as a complication is of interest. The obvious thought in the association of tuberculosis with empyema is that a tuberculous pleural effusion or empyema had become secondarily infected, but this occurred in only one case which was also the only fatality attributable to tuberculosis. In the other five cases tuberculosis appeared in a sinus tract of long standing through which acute empyema had been drained. In four of the cases no involvement of the lung could be demonstrated. The other case is worthy of a brief summary.

The patient was a 7 year old girl admitted with pneumonia. On admission a Mantoux test was negative. In due course she developed empyema from which pneumococcus Type 1 was obtained. The pus became too thick to aspirate and, as she was critically ill, intercostal closed drainage with suction and irrigation was established. The twentieth day after operation she had a positive pneumococcus Type 1 blood culture although the empyema seemed well drained. Her condition than began to improve slowly but ten weeks after operation an undrained pleural pocket was opened by resecting three ribs. It communicated with a bronchopleural fistula. Next she developed varicella and was transferred to Willard Parker Hospital (for contagious diseases). Upon her return the sinus was still open so a third operation was done in which two more ribs were resected and granulation tissue removed. Section of the latter showed it to be tuberculous and now the Mantoux was positive. Some time later tuberculosis was found in the contralateral lung and it was thought that she had a spread from the granulation tissue by way of the bronchial fistula. When last seen she had active pulmonary tuberculosis, an open chest sinus and tuberculous iritis and keratitis.

This is a rather extreme example of the damage which can be caused by a bronchopleural fistula. Ordinarily it will close spontaneously and often its presence is discovered quite by chance.

One of our patients had an unsuspected bronchopleural fistula and died of shock when Dakin's solution which was being used for irrigation was aspirated into the lung through the open fistula. Carlson and Bowers report another similar case of sudden death from the use of Dakin's solution in the presence of an unsuspected bronchopleural fistula; and J. H. Heyl has told me of a third. For this reason we seldom use irrigation in our empyema cases. On the other hand a bronchopleural fistula in the presence of a small empyema may permit sufficient drainage to bring about a spontaneous cure of the empyema without operation.

Bronchopleural fistula occurred thirty-one times and was associated with a fatal outcome in seven instances. In four of these fatal cases the bronchopleural fistula was of secondary importance to other and more severe complications. Another fatality has just been mentioned and in the other two the bronchopleural fistula caused a tension pneumothorax, the most dangerous effect of this complication.

Tension pneumothorax occurs when the fistula breaks through in such a way that a valve is formed permitting the air to enter the pleural cavity on inspiration but blocking its exit. This causes an abrupt collapse of the lung and shifting of the mediastinum with consequent embarrassment of the heart action. The patient suddenly becomes dyspneic; his pulse becomes fast and weak; and he may be cyanotic. If the positive intrapleural pressure is allowed to build up, death soon takes place from cardiac embarrassment. Not infrequently, however, in the older children the increased pressure closes the valve. Both our fatalities occurred in infants, one of eleven months and the other of fourteen months.

Immediate relief of the positive pressure must be obtained. This can most quickly be accomplished by aspiration of the air through a needle which may be left in or inserted again if the pressure rebuilds. If relief is not thus readily obtained inter-

costal drainage through a flapper tube or closed system is indicated.

One of our cases was that of a 4 year old girl who had undergone intercostal drainage for an encapsulated empyema. On her eighth postoperative day she suddenly developed dyspnea, cyanosis and rapid, thready pulse. She was in a critical condition for a short time and then began to improve. Because her empyema had already been drained, tension pneumothorax was not immediately considered and when it was discovered was found to originate from a bronchopleural fistula at a distance from the walled-off empyema. This eventually had to be treated by rib resection and packing whereupon the fistula closed spontaneously.

When these measures fail to close the fistula it may be due to a thickened pleura preventing expansion of the lung. Decortication of the lung may then be necessary and if this is not sufficient the fistula will have to be closed by a muscle-flap transplant.

The thickened pleura prevents expansion of the lung and produces chronic empyema, a complication occurring twelve times in this series. Decortication of the lung to permit expansion is usually all that is necessary although this may have to be combined with a thoracoplasty to close the dead space. The longer a chronic empyema exists the more will have to be done to correct it and it is therefore advisable to operate as soon as it becomes apparent that the lung will not expand to the chest wall.

Most of the chronic empyema can be avoided. Delay in operating is one preventable cause but this is less of a factor than inadequate drainage. Apparently a lung can stand collapse from a large collection of undrained pus without impairing its ultimate power of expansion much longer than it can a collapse due to inadequate drainage. It therefore behooves the surgeon to watch the drainage and the closure of the cavity and not to be led astray by a normal temperature.

Cellulitis of the chest wall is mentioned by various authors as an important complication but occurred in our cases but four times. In one case it was associated with erysipelas, streptococcus septicemia and pneumonia, which brought about a fatal result. In the others it was due to an anaerobic infection along the tract of an aspirating needle in a putrid empyema. When a putrid empyema is suspected aspiration should not be done until all is in readiness to do a thoracotomy should the suspicions be realized.

Perforation of the diaphragm occurred three times. Twice it was due to a subphrenic abscess which perforated upwards to form the empyema and in the other case to tuberculosis.

Two cases were complicated by the presence of foreign bodies in the chest. In one instance a rubber dam drain slipped into the chest in one of our own cases (an accident of which we were aware) and required a secondary operation to recover it. The other case was that of a child who had been operated upon for empyema in his home three years before. Because it is so easy to lose drainage material in the chest, surgeons are on their guard and the accident is relatively infrequent.

Another accidental case of empyema is worth recording. It followed bronchoscopy for the purpose of removing a peanut which had become lodged in a bronchus of a 7 year old boy. While the instrument was in place he struggled and the trachea was perforated. Subcutaneous emphysema followed at once, necessitating tracheotomy. Pyopneumothorax developed and was treated by intercostal drainage but lung abscess further complicated the situation and resulted in death.

The remaining miscellaneous complications include nephritis, osteomyelitis of the tibia, suppurative arthritis, tonsillitis, carbuncle and multiple lung abscesses.

CONCLUSIONS

The fatalities of empyema are due to the complications which are, for the most part, the result of the original infection or of some intercurrent infection.

The nonfatal complications are of a similar nature and also arise from the surgery. Some of the surgical complications can be prevented.

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COLON bacilli frequently gain access to one or the other kidney by the close lymphatic connections of the colon with the kidneys, of the caecum and ascending colon with the right kidney, and of the descending colon and sigmoid with the left kidney. Urinary infection is common in ulcerative colitis, polyposis, diverticulitis, etc.
From—"Convalescent Care" (New York Academy of Medicine).

AN EVALUATION OF SORBITOL AS A DEHYDRATING AGENT*

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SINCE Weed and McKibben[1] demonstrated that the cerebrospinal fluid pressure could be reduced by the intravenous administration of hypertonic solutions of dextrose, sodium chloride, sodium sulfate and sodium bicarbonate, there has been an extensive application of this principle. The respiratory and cardiac disturbances observed in experimental animals following the intravenous injection of the sodium salts, as well as clinical experiences, militated against the use of these chemicals in the human. Further observations indicated that hypertonic solutions of dextrose given intravenously were attended by few untoward reactions and accordingly this chemical was employed extensively for the reduction of intracranial tension. There were, however, some undesirable features associated with the use of this carbohydrate, one in particular being that the reduction in cerebrospinal fluid pressure lasted for a relatively short period and not infrequently was followed by a secondary rise in pressure to a level well above that observed prior to the injection.

In the quest for a substance that would produce a prolonged reduction in pressure without undesirable by-effects, sucrose was investigated both experimentally[2] and clinically.[3,4] A 50 per cent solution of this chemical was found to be more effective, in that the reduction in cerebrospinal fluid pressure was commonly of longer duration than that observed following the injection of a comparable quantity and concentration of dextrose, yet the results were not uniformly satisfactory.

Recently another hypertonic solution, sorbitol, has been offered for clinical use as an osmotic dehydrating agent and diuretic. This substance is reported to be a modified monosaccharide, $C_6H_{14}O_6$, derived from the reduction of dextrose or levulose. It appears not to be hydrolyzed to any extent when injected intravenously in the human and is rapidly excreted in the urine. In 50 per cent solution sorbitol possesses the high osmotic pressure of dextrose of the same strength and almost twice that of a 50 per cent solution of sucrose. Burget, Todd and West[5] injected large doses of 50 per cent solution of sorbitol intravenously into dogs without evidence of toxicity. These authors reported that the diuresis produced by this chemical was greater than that observed following the administration of an equal quantity of 50 per cent solution of sucrose.

The object of the present investigation was to determine the effect of sorbitol on the cerebrospinal fluid pressure, pulse, respiration, blood pressure and psychologic state in human subjects with and without disease of the central nervous system. Thirty-three patients with various diseases of the intracranial structures were studied, with five patients as controls who had neither clinical evidence of a pathologic process implicating the nervous system nor increase in the intracranial tension as measured by the cerebrospinal fluid pressure. The age of the patients ranged from 23 to 86 years and the male to female ratio was 4:1. Ten detailed studies concerning the cerebrospinal fluid pressure were carried out on nine patients. Clinical observations were made following each of the forty injections given to the remaining twenty-nine patients.

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METHOD

For the detailed studies, the patient was placed in a lateral position with the cerebrospinal axis on a horizontal plane. The cuff of a sphygmomanometer was applied to the arm of the uppermost side and a stethoscope led away from the antecubital fossa. A 17-gauge spinal needle attached to a mercury manometer was introduced into the lumbar thecal sac without external loss of cerebrospinal fluid and the zero reading of the manometer placed at the level of the needle. As a control in each study, the mensuration of the cerebrospinal fluid pressure, blood pressure, pulse rate, and respiratory rate were recorded every five minutes throughout periods ranging from twenty to sixty minutes or until a constant level of all these factors was maintained. An injection of 50 c.c. of 50 per cent solution of sorbitol was then given into the antecubital vein of the arm of the lowermost side. The period of the injection of sorbitol was ten minutes in seven instances and forty minutes in the remaining three. The cerebrospinal fluid pressure readings were recorded every minute during the period of injection and every two and one-half minutes during the remainder of the observations, while the blood pressure readings, pulse rate and respiratory rate were noted every ten minutes.

For the clinical studies the patients were in the supine position. Observations concerning the blood pressure, pulse rate, respiratory rate, temperature, and the state of consciousness were recorded, then 50 c.c. of 50 per cent solution of sorbitol were administered intravenously. During the subsequent six to eight hours these vital signs were determined and recorded at thirty minute intervals. The actual period of injection was ten minutes in seven instances and the remaining thirty-three injections were given slowly over periods ranging from thirty to forty minutes.

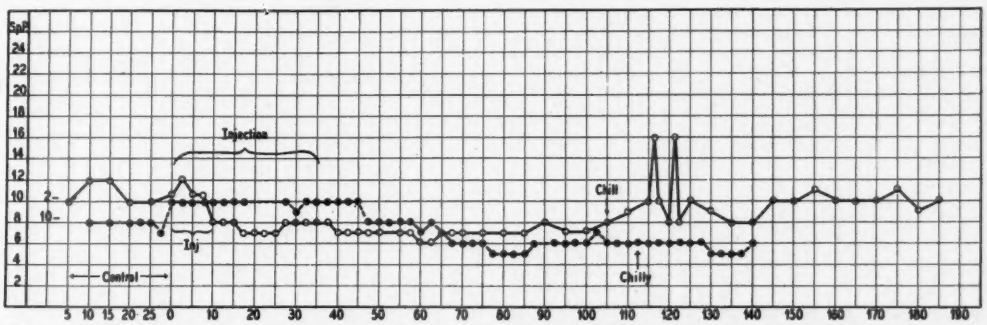

FIG. 1. Chart showing the cerebrospinal fluid pressure (S_pP) in Cases II and X (normal controls). The period of injection of sorbitol was ten minutes in Case II (open circles) and thirty-five minutes in Case X (solid circles). Time recorded in minutes.

ABSTRACTS OF CASE HISTORIES USED IN THE DETAILED STUDIES

CASE I. J. M., a 48 year old male, had had a craniotomy four and one-half months previously with subtotal removal of a right parietal glioma. The scalp over the cranial opening was moderately tense. During the period of control observations, the cerebrospinal fluid pressure was 17 mm. Hg., pulse 110 per minute, blood pressure 114/70 mm. Hg., and the respirations 36 per minute. Following the intravenous injection of 50 c.c. of sorbitol in eleven minutes, there was a gradual fall in cerebrospinal fluid pressure to 14 mm. Hg. The pressure was maintained at this level for one hour and fifty minutes at which time the observations were discontinued because of a severe chill which had been present for thirty minutes. There was no essential change in the vital signs prior to the chill. The area of decompression was less tense after the injection and the patient seemed somewhat more alert.

CASE II. E. D. V., a normal 38 year old male, was a control subject. The cerebrospinal fluid pressure was 10 mm. Hg., pulse rate 73 per minute, blood pressure 116/76 mm. Hg., and respirations 20 per minute. An injection of

50 c.c. of sorbitol was given over a period of ten minutes, resulting in an immediate fall in cerebrospinal fluid pressure to 7 mm. Hg.

Case iv. J. L., a 28 year old male complaining of severe intermittent headache of six months' duration, was conscious and alert.

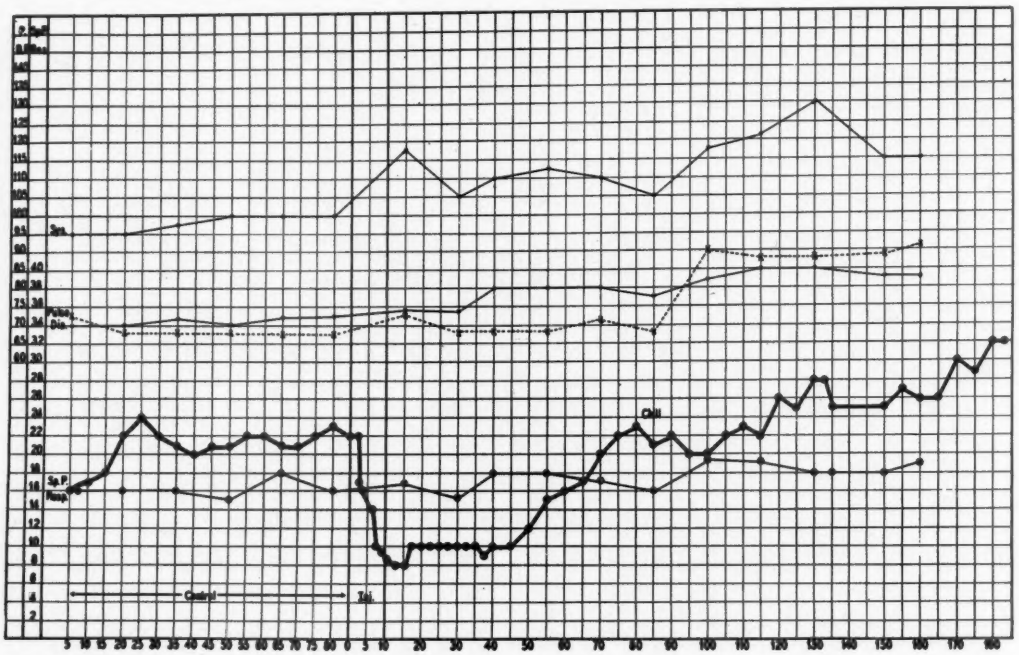

Fig. 2. Case v (brain tumor) showing the prompt reduction in cerebrospinal fluid pressure (S_pP) following the injection of sorbitol, with subsequent secondary rise in pressure above the original level. Time recorded in minutes.

(Fig. 1.) There was no change in the pulse rate or blood pressure until a chill lasting thirty minutes occurred one and one-half hours after the injection, at which time the pulse rate increased to 96 per minute. There was no alteration in the state of consciousness during the period of observation.

Case iii. E. K., a 43 year old male, had sustained a severe brain injury and was alternately stuporous and restive at the time of observation, forty-four hours after injury. A right hemiparesis was demonstrable and seemingly a speech disturbance was present. The cerebrospinal fluid was contaminated with blood and the pressure was 10 mm. Hg. The pulse rate was 68 per minute, blood pressure 90/54 mm. Hg., and respirations 20 per minute. Following the intravenous injection of 50 c.c. of sorbitol in ten minutes, there was a prompt reduction in cerebrospinal fluid pressure to 4 mm. Hg. with a rise to 6 mm. Hg. at the end of forty-five minutes. It became necessary to discontinue the observations at this time because of marked psychomotor acceleration. At the conclusion of the observations the pulse rate was 74 per minute and the blood pressure and 106/70 mm. Hg.

An intramedullary mesencephalic lesion, presumably tumor, had been demonstrated by ventriculography on a previous admission. The observations are recorded in Figure 2. About one and one-half hours after the injection of 50 c.c. of sorbitol there was a chill lasting nine minutes. The state of consciousness remained unaltered.

Case v. L. G., a 62 year old male, was moderately drowsy and presented clinical evidence of an intracranial neoplasm. Subsequent craniotomy verified a left frontotemporal metastatic tumor. The cerebrospinal fluid pressure was 40 mm. Hg., pulse rate 120 per minute, blood pressure 152/98 mm. Hg., and respirations 28 per minute. The intravenous injection of 50 c.c. of sorbitol in ten minutes reduced the cerebrospinal fluid pressure to 24 mm. Hg. where it remained for twenty minutes and then gradually returned to the control level, one hour after the completion of the infusion. One and one-half hours after the injection, the cerebrospinal fluid pressure was 56 mm. Hg. A chill lasting twenty minutes occurred at this time. The observations were concluded three hours after the injection, and at the time, the cerebrospinal fluid pressure was 40 mm. Hg.,

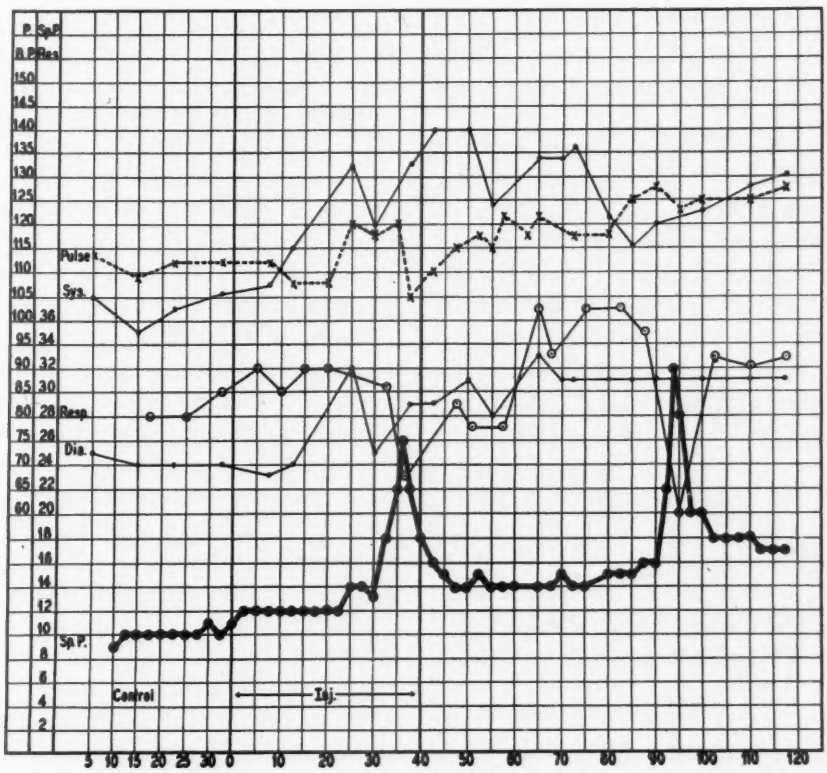

Fig. 3. Case ix (craniocerebral trauma), showing a progressive rise in cerebrospinal fluid pressure (S_pP) following the administration of sorbitol and the associated alterations in the vital signs. Time recorded in minutes.

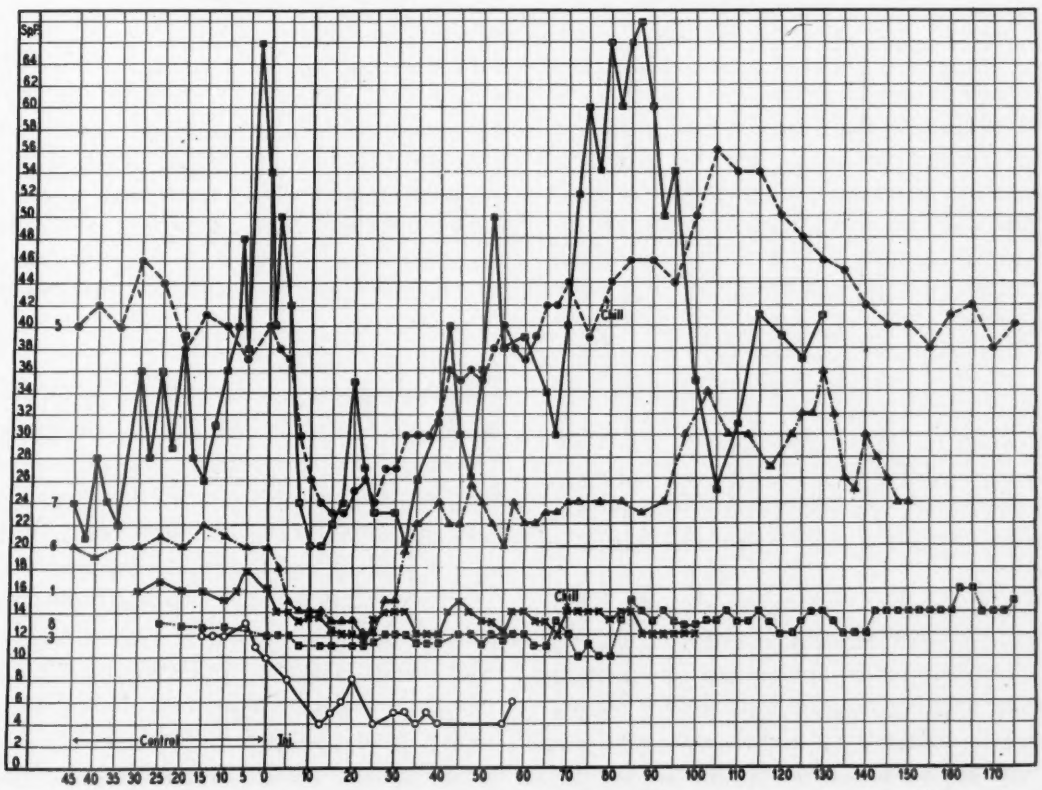

Fig. 4. Composite chart of the cerebrospinal fluid pressures in Cases i, iii, v, vi and vii, illustrating the variable effects following the injection of sorbitol. Note the marked spontaneous fluctuations in pressure in Case vii particularly. Time recorded in minutes.

pulse rate 130 per minute, blood pressure 154/106 mm. Hg. and respirations 30 per minute. There was no marked change in the state of consciousness.

Case vi. The same patient used for the study recorded in Case v. The control readings established the cerebrospinal fluid pressure at 20 mm. Hg., pulse rate 90 per minute, blood pressure 158/98 mm. Hg., and respirations 22 per minute. The intravenous injection of 50 c.c. of sorbitol in eight minutes, reduced the cerebrospinal fluid pressure to 12 mm. Hg., but within forty-five minutes the pressure returned to the original level. A secondary rise in pressure to 34 mm. Hg. occurred one and one-half hours after completion of the infusion. At the conclusion of the observations two and a half hours after the injection the pressure was 26 mm. Hg. The blood pressure, pulse readings, and respirations remained essentially unchanged and the patient seemed somewhat more responsive.

Case vii. H. S., a 48 year old male complaining of severe headache, was alternately drowsy and restive. Craniotomy performed subsequent to the study verified a large right frontal glioma. Although the patient was quiet and the blood pressure, pulse rate, and respirations remained constant during the control period of forty-five minutes, the cerebrospinal fluid pressure fluctuated between 40 and 60 mm. Hg. Following the intravenous injection of 50 c.c. of sorbitol during ten minutes, there was a reduction in the cerebrospinal fluid pressure to 20 mm. Hg. at which level it remained for about twenty minutes. There were no significant changes in the pulse rate, blood pressure or respirations. A secondary rise in pressure to 70 mm. Hg. occurred one hour after completion of the injection. At the conclusion of the observations, two hours after the injection, the cerebrospinal fluid pressure was 40 mm. Hg., pulse rate 50 per minute, blood pressure 106/60 mm. Hg., and respirations 14 per minute. There was no change in the state of consciousness.

Case viii. J. N., a 38 year old male, had a large left parietotemporal cranial defect, the result of a craniotomy performed six months previously with subtotal removal of a glioma. The patient complained of headache and was mildly drowsy at the time of this study. The control observations established the cerebrospinal fluid pressure at 12 mm. Hg., pulse rate 66 per minute and blood pressure 118/22 mm. Hg. During the infusion of 50 c.c. of sorbitol over a period of forty minutes, there was no essential change in the cerebrospinal fluid pressure. Two hours after the injection, the cerebrospinal fluid pressure was 14 mm. Hg., and remained at this level until the conclusion of the observations, three hours after the administration of the sorbitol. The headache was somewhat alleviated by the medication.

Case ix. J. W., a 60 year old male, had sustained a severe craniocerebral injury eight hours prior to the beginning of the study. The patient was stuporous but would resist painful stimuli. No gross paralysis of the extremities could be demonstrated. The cerebrospinal fluid was contaminated with blood. During the intravenous infusion of 50 c.c. of sorbitol over a period of forty minutes, there was rise in the cerebrospinal fluid pressure, associated with irregular respirations. The observations are recorded in Figure 3. A mild chill lasting eight minutes occurred one and a quarter hours after the injection. The stupor appeared to be more profound at the conclusion of the observations.

Case x. J. S., a healthy 32 year old male, without demonstrable evidence of disease of the central nervous system, was a control subject. An injection of 50 c.c. of sorbitol was given over a period of forty minutes. The cerebrospinal fluid pressure is recorded in Figure 1. The pulse rate, blood pressure and respiratory rate maintained a normal level during the observations. A chilly sensation with "gooseflesh" over the body, lasting twelve minutes, occurred two hours after the infusion. There was no change in the state of consciousness.

CLINICAL STUDIES

In this group twenty-nine patients were used as subjects in forty observations. Three patients without evidence of central nervous system disease and four with brain tumor (preoperative) were each given a single injection of sorbitol; three patients (postoperative brain tumor) with large cranial defects were given a total of seven injections; twelve patients with craniocerebral trauma received fourteen injections; two patients with postoperative brain edema three injections; five patients with miscellaneous disorders, including post-traumatic syndrome, were given a

total of nine injections. The three patients used as controls showed no obvious changes in the psychologic or constitutional state following the injection, aside from chills with an accompanying acceleration of pulse. Improvement, as evidenced by a more lucid state of consciousness, relief of headache and frequently a disappearance of some of the abnormal neurologic findings, was recorded in twenty of the thirty-seven observations. The degree of improvement was not uniform in all types of cases as indicated in Table 1. A severe chill, lasting

Table 1

Type of Case	Percentage Improved
Brain tumor (preoperative)	50
Brain tumor with decompression (postoperative)	14
Craniocerebral trauma (acute)	57
Brain edema (postoperative)	66
All others (post-traumatic syndrome)	77

from three to thirty minutes, followed by a febrile rise to 103 or 104°F., occurred after thirteen injections while mild reactions characterized by chilly sensations, "goose-flesh," and a mild febrile rise followed six injections. There were no significant changes in pulse rate, blood pressure, respiratory rate or temperature in instances not associated with a chill. Mild to moderate cyanosis of the mucosa and nail beds was noted on several occasions after the injection, particularly in the cases of craniocerebral injury.

DISCUSSION

The intravenous injection of sorbitol in each of the two patients used as controls (Cases II and X) in the detailed studies resulted in a mild reduction of cerebrospinal fluid pressure in both instances. In one, the reduction was sustained for 140 minutes (observations discontinued at this time) and in the other for 120 minutes followed by a return of the pressure to the pre-injection level where it remained until the conclusion of observations (190 minutes after injection). Similar results have been observed following the injection of both dextrose and sucrose. For example, in Case II an injection of 100 c.c. of 50 per cent solution of dextrose effected a reduction in cerebrospinal fluid pressure that was sustained for 220 minutes (observations concluded at this time). An injection of 100 c.c. of a 50 per cent solution of sucrose given forty-eight hours later did not appreciably alter the cerebrospinal fluid pressure during a period of observation of 190 minutes.

The reduction in cerebrospinal fluid pressure observed by Weed in the animal experiments following the administration of dehydrating agents is, in general, comparable to that observed by us in the normal human. Weed reported an individual tolerance of each animal so that, consequently, the effects of a drug could not be predicted in advance. We have found the same to be true in the human.

This inconstancy of behavior in the cerebrospinal fluid pressure that may follow the injection of a dehydrating agent is even more marked in instances associated with increased intracranial tension. A comparison of Figures 1 and 4 emphasizes this fact. The latter depicts the cerebrospinal fluid pressure in six patients presenting evidence of varied intracranial pathologic processes. Rapid spontaneous fluctuations in pressure ranging from 20 to 40 mm. Hg. were noted in several instances during the period of control observations. This phenomenon usually disappeared during the period of reduced pressure produced by the injection of sorbitol but promptly recurred when the cerebrospinal fluid pressure returned to or exceeded the original level. Frequently a complete oscillation in pressure has been observed in five seconds. There was no physical effort on the part of any of the patients nor significant change in the vital signs associated with this phenomenon. Many of the patients in whom this was observed complained of sudden severe headache during the rise in cerebrospinal fluid pressure and relief was experienced as the pressure receded.

In three instances (Cases I, VI and VIII), there was clinical improvement following

the injection of the sorbitol and in each experiment there was a reduction in cerebrospinal fluid pressure with a minimal secondary rise, the final pressure remaining near the original level. In four of the studies, (Cases IV, V, VII and IX), improvement was not observed. In three of these there was a reduction in cerebrospinal fluid pressure followed by a marked secondary rise, the pressure at the conclusion of the observations remaining higher than the pre-injection level. In the fourth, a patient with a severe craniocerebral trauma, there was an immediate progressive rise in cerebrospinal fluid pressure following the injection and at the conclusion of the study the pressure was more than twice the original level. (Fig. 3.) A similar effect following the intravenous injection of 50 per cent solution of dextrose has been previously reported by one of us[6] and again it should be stressed that the administration of dehydrating agents during the first twenty-four to forty-eight hours following severe brain trauma is not without danger.

In five of the patients in this series, studies had previously been conducted utilizing 100 c.c. of 50 per cent solution of dextrose and an equal quantity and concentration of sucrose. The 100 c.c. dose was used as a standard in these observations while the recommended dosage of 50 c.c. of sorbitol was used in the present study. The changes in the cerebrospinal fluid pressure following the intravenous injection of 50 c.c. of sorbitol were similar to those observed following the administration of 100 c.c. of dextrose, there being an initial reduction in pressure followed by a marked secondary rise in three of the five cases. The reduction in pressure effected by the injection of 100 c.c. of sucrose in the same patients was not associated with a secondary rise. There were no significant disparities in the changes effected in the cerebrospinal fluid pressure following the injection of sorbitol, dextrose, or sucrose in the remaining two cases.

A severe chill associated with a febrile rise to 103 or 104°F., occurred in four instances and chilly sensations in two additional cases in the ten detailed studies. In the entire series of fifty injections, a chill occurred in seventeen and chilly sensations were reported in eight additional instances, making a total of twenty-five, or an incidence of 50 per cent. Chills were observed less frequently in those patients in whom a relatively normal intracranial tension was recorded. The rate of injection seemed to be a factor in the production of this reaction, there being an incidence of 64 per cent following the rapid injection technique (ten minutes) in comparison with 40 per cent after the drip method (thirty to forty minutes). One patient (Case I) was given four injections of sorbitol, one by the rapid and three by the drip method, and following each there was a severe chill. In this individual, previous injections of 100 c.c. doses of 50 per cent solution of both dextrose and sucrose had been unattended by a chill. It is worthy of note that the severe chills observed during the detailed studies did not materially influence the cerebrospinal fluid pressure except in Case II as recorded in Figure 1. In this instance during the height of the chill, a rapid rise and fall in pressure occurred twice in five minutes.

TABLE II
CLASSIFICATION OF CASE STUDIES WITH DISTRIBUTION OF REACTIONS

	Injections	Chills	Mild Reaction
Controls	5	1	1
Brain tumor (preoperative)	8	3	0
Brain tumor with decompression (postoperative)	9	5	1
Brain trauma (acute)	16	5	3
Post-traumatic syndrome, etc.	8	1	2
Postoperative brain edema	3	2	1
Postencephalography	1	0	0
	50	17	8

SUMMARY AND CONCLUSIONS

A total of fifty intravenous injections of sorbitol was given to thirty-eight patients representing a variety of intracranial lesions. Clinical improvement as evidenced by a more lucid state of consciousness and relief of headache was noted in 54 per cent. Detailed observations concerning the cerebrospinal fluid pressure, pulse, blood pressure, respirations and psychologic state were recorded in ten experiments. Included in the detailed studies were two patients without evidence of central nervous system disease. In each experiment on the two subjects a slight reduction in cerebrospinal fluid pressure was effected and sustained for over two hours. In four of the five patients in whom an elevation in cerebrospinal fluid tension was demonstrated, the injection of sorbitol produced an immediate reduction in the pressure, followed by a secondary rise to a level higher than that observed prior to the injection. In a patient who had sustained a recent craniocerebral trauma there was an immediate progressive rise in cerebrospinal fluid pressure to twice the original level following the injection.

In general, the action of sorbitol as measured by the reduction in cerebrospinal fluid pressure is comparable to dextrose but less satisfactory than sucrose. The frequent occurrence of chills following the intravenous injection of this chemical militates against its use as a dehydrating agent.

The sorbitol utilized in this study was donated by Abbott Laboratories.

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MESENTERIC VENOUS OCCLUSION: A CLINICAL ENTITY

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THERE have been about 772 cases of mesenteric vascular occlusion reported and discussed in the literature but most of the writings concern arterial occlusion. The author believes with Donaldson and Stout, and with Warren and Eberhard that venous occlusion is a separate clinical entity and should be treated as such in the medical literature. The aim of this paper is to present additional data on the clinical picture of mesenteric venous occlusion to make this lesion more clear cut.

ANATOMY

Anatomically, the venous and arterial systems of the mesentery are quite different. The arterial system is the more simple of the two groups of vessels, with the superior mesenteric artery arising from the aorta at an acute angle immediately below the celiac axis and its main stem paralleling to some extent the course of the abdominal aorta. Thus it may be seen that the superior mesenteric artery has a direct connection with the heart and thereby is vulnerable to emboli from above. In contrast to the arterial distribution all blood collected in the veins of the abdominal part of the digestive tract (except the lower part of the rectum), spleen, pancreas, and gall-bladder passes into the portal vein by which it is filtered through the liver, then through the hepatic veins to the vena cava. The liver in this system of veins may be compared to a dam with spillways between two large reservoirs of venous blood. Any thrombus arising in the liver may pass on as an embolus into the cephalad channels, producing damage where it lodges. An embolus, as such, could not pass into the caudad channels against the current of venous blood, but a thrombus arising in the liver or below could block off the venous channels, resulting in a descending thrombosis extending into part or all of the branches of the superior mesenteric vein. In contradistinction, a thrombus arising in the smaller radicles of this vein may give rise to an ascending thrombosis or become embolic to produce infarction of the liver. The anatomic factors of the venous system predispose to thrombosis more than those same factors do in the arterial system.

PATHOLOGY

Virchow reported the first case of vascular occlusion of the superior mesenteric artery and later studies were made by Jackson, Porter and Quinby (1904) and Trotter (1913). The pathologic result of the occlusion in the intestine has been fairly well established, while the initiating pathologic states are not agreed upon entirely. Warren and Eberhard, in quoting Sprengel, stated that either arterial or venous occlusion is due to sudden embolism, while venous occlusion results from a slower progressive thrombosis, but that the difference in speed of events may not be obvious clinically until the compensatory powers of the body are overcome by a failing circulation. Typical acute symptoms then appear without warning. In addition to physiologic changes in the intestinal tract and its blood vessels, Donaldson and Stout considered such lesions as intussusception, volvulus, general debility of acute infections, trauma, acute and chronic enteritis and infective or other emboli as initiating pathologic states in venous thrombosis.

Whittaker and Pemberton found 60 per cent of sixty cases unrelated to previous operations while 40 per cent definitely followed surgery. Thrombosis occurred more frequently in surgical patients with lesions predisposing to thrombus formation such as splenic anemia and arteriosclerosis. These authors considered that mesenteric venous occlusion occurs secondary to a

descending thrombosis from hepatic and portal lesions. Associated portal thrombosis was found in 30 per cent of the cases and pulmonary thrombosis in 11.6 per cent. Previous attacks are indicated by finding old organized and non-occlusive clots in a few cases. Voigt stated that a poor prognosis of appendicitis in older persons may be due to mesenteritis, lowered resistance and degenerative diseases predisposing to thrombosis. Feldstein and Goldstein reported a case involving the superior mesenteric vessels due to prolonged suppuration of the mastoid. Skinkawa and Mari mentioned a case caused by calcification of a mesenteric lymph node. Boyce and McFetridge found that the origin and evidence of occlusions cannot always be determined.

The immediate gross pathology resulting from venous occlusion is variable. The involved bowel segment becomes dusky and dark red in color with loss of sheen. The intestinal wall is edematous and thickened, while beneath the serosa a gelatinous appearance exists. The mesentery is thickened and edematous with loss of mobility and there are palpable nodular areas (due to thrombi within the vessels) in the involved portion. The mesentery may be pale or present an extension of the dusky red color from the intestine, but any discoloration may be hidden by mesenteric fat. The involved mucous membrane is red, hemorrhagic, and swollen, not showing ulceration. Douglas considered that color alone is insufficient to determine viability but that escaping blood from a surface pinprick and careful observation of circulation may help determine the extent of resection necessary. Lower and Glazer, by microscopic section, showed the involved bowel and mesentery in their case to have extensive edema, venous engorgement, and extravasation but no ulceration of the mucous membrane.

ETIOLOGY

In reviewing the literature one is impressed by the multiplicity of initiating pathologic lesions ascribed to the causation of venous and arterial occlusion. Practically all of them, however, can be classified under the following main divisions:

1. Debilitating and degenerative diseases.
2. Inflammatory lesions of the abdomen.
3. Neoplastic diseases of the abdomen.
4. Mechanical factors (surgery, trauma, obstruction and hernia).
5. Undetermined origin of the thrombosis.

Oschner stated that an insufficiently appreciated cause of mesenteric venous occlusion is associated with prolonged ingestion of alcohol and that the prognosis in this type is good. He reported five such cases, all of which were operated upon and only one resected, with the recovery in those not resected. Larson found that venous thrombosis most often resulted from abdominal septic processes; 31 per cent followed appendicitis and twenty-five were associated with liver disease. Green wrote that fatigue and prolonged emotional strain may cause venous occlusion, following a study of his cases and the experiments of Cannon, showing that increased adrenin due to these factors decreased clotting time of the blood to one-half. Brady stated that if a patient with cirrhosis of the liver or endocarditis suddenly develops an acute abdominal condition, one may suspect mesenteric thrombosis.

ADDITIONAL STATISTICS

This study was made on 101 cases of venous occlusion taken from the literature, two from the Guthrie Clinic, and one from the author's experience. The source of the cases is shown in Table 1.

It was found that of 100 patients of known age, the youngest was four months and the oldest 76 years. It is obvious that no age is excempt from this catastrophe. (Table 11.) The average for all patients was 43.1 years, while the age range for the greatest incidence was 21 to 70 years. The decade of life with the greatest incidence was 50 to 60 years. When these figures are interpreted, venous occlusion occurs most

often at the age in which degenerative diseases begin to make their appearance.

Table I

Author	No. of Cases	Deaths	Recoveries
Brady	7	5	2
Larson	16	16	0
Douglas	1	0	1
Lower and McCleery	1	0	1
Wood	4	4	0
Pollock	1	0	1
Mallory	1	0	1
Mathews	1	0	1
Milch and Masotti	1	1	0
Donaldson and Stout	3	3	0
Warren and Eberhard	65	39	26
Guthrie Clinic	2	2	0
Author's case	1	1	0
Totals	104	71	33

Table II

Ages	No. of Cases
0–10	2
11–20	7
21–30	18
31–40	18
41–50	17
51–60	24
61–70	13
71–80	1

An attempt was made to correlate the length of time of illness before the patient came to surgery, with recovery or death, in order to show some prognostic index. There were nineteen recoveries out of a total of thirty-three patients who had been ill for an average of 13.5 days before operation. Fourteen stated that the onset was sudden and these were presumably operated on without delay. It will be seen that 57.5 per cent of the patients recovering had been ill for nearly two weeks. This does not support the calculations of Warren and Eberhard that the mortality rate increases from 25 per cent in the first twelve hours to 83.3 per cent after four days. Among the seventy-one deaths in this series of 104 cases, forty-six patients had an average preoperative illness of 15.4 days; nine patients claimed sudden onset of the illness; three deaths followed surgery for other conditions; six patients were ill from a few to several days; in four cases there were no data; and one patient claimed illness for several years.

In this survey it was found that associated pathology occurred in a number of these 104 cases. Although the records may be incomplete from this standpoint, appendicitis occurred nineteen times (17.7 per cent) and was considered the initiating cause of the vascular accident. Portal thrombosis occurred sixteen times, to account for an equal number of occlusions, while diseases of the liver, with or without splenic and pancreatic lesions, contributed to the initiating causes of thrombosis in a number of patients. Blood dyscrasias (polycythemia vera) accounted for the thrombosis of the mesenteric veins in three patients. Pelvic inflammatory disease may contribute to the etiology of venous occlusion, but of all such cases it is uncommonly associated with thrombus formation in the mesenteric vessels. Acute phlebitis in peripheral veins may cause the mesenteric lesions. Other degenerative diseases may cause venous occlusion. It was found in this survey that 99 per cent of the occlusions occurred in the superior mesenteric vein, while only the patients with this vein alone involved recovered from the accident. Patients having hepatic, splenic, pancreatic or portal involvement invariably died, and those without any associated lesions to account for the thrombosis were most likely to survive.

An attempt was made to determine the relationship of the extent of the involved mesentery and intestine to the ultimate outcome of the patient. There were twenty-two out of the thirty-three patients who recovered in whom only the ileum was involved, while five involved the jejunum alone, and in four a portion of both these segments showed the effects of the occlusion. In one patient, the veins were occluded, but it was stated that the bowel was not involved. Still another was reported to have the entire jejunum and ileum involved; this patient survived after

a resection. Flint, as quoted by Boyce and McFetridge, showed that 50 per cent of the small bowel may be removed with recovery and normal function.

Among the seventy-one who died, twenty-two had the ileum alone involved and two involved only the jejunum; fifteen showed combined involvement of the ileum and jejunum; five involved the lower ileum and cecum; and in five some part of the colon was attacked. There were twenty-two patients in whom the small bowel was indicated as being involved but no further localization was noted. Eight patients who died from venous occlusion had the entire small bowel affected.

The thirty patients in the recovery group whose ages are known averaged 38.9 years, whereas the sixty-eight who died had a mean age of 44.4 years. This would indicate to some extent that the younger the patients are, the better the prognosis should be.

The average length of the bowel involved in twenty-two of thirty-three patients recovering was 38.1 inches. When this is compared with thirty-seven patients who died, having an average of 102.4 inches of the intestine infarcted, there is definite evidence that the prognosis depends on the extent and location of the lesion. Roughly, from these figures, it may be concluded that if no more than 36 to 38 inches of the small intestine shows venous occlusion, the prognosis is hopeful, while an amount greater than this causes the mortality rate to rise rapidly.

The general mortality rate of this series of 104 cases was 68.2 per cent. This is better than the 85 to 95 per cent commonly stated in the literature for the arterial and venous cases combined.

SYMPTOMATOLOGY

There is a fairly definite symptom pattern as demonstrated by the case reports and indicated by other writers on this subject. It is generally conceded that the symptoms of arterial occlusion are more fulminating while the progression in the venous type is slower. The onset of symptoms in venous occlusion may mean an unfavorable delay for the patient and may be misleading to the diagnostician. The symptoms, physical findings, and the laboratory findings are discussed separately.

Pain. The onset of pain may be rapid or slow. It may be colicky in character with a disproportion of abdominal tenderness and duration of symptoms to rigidity, according to Donaldson and Stout. Whittaker and Pemberton stated that the severe pain of venous occlusion is preceded by a less severe dull ache which is more frequently constant than intermittent. All authors agree that pain is the presenting symptom. The patient of Lower and Glazer was sent to the clinic for a urologic study because the pain suggested renal colic. It was found to be 100 per cent present in the cases studied by Meyer, but he did not differentiate the pain of arterial and venous occlusion. Dunphy found that abdominal vascular pain was the result of an anoxemia of the intestinal wall and that it was a true visceral pain manifested through sensory neurones in the sympathetic nerves independently of the musculocutaneous pathways. The contrast between the severity and persistence of the pain and the paucity of physical findings was the most significant diagnostic feature of mesenteric venous occlusion.

Vomiting. Whittaker and Pemberton write that vomiting is more frequent in arterial occlusion, and that in 50 per cent of the combined venous and arterial cases vomiting occurred. The pure venous lesion should present this symptom less frequently. Vomiting did not occur in the case reported by Mallory in which 3 or 4 feet of bowel was resected, with recovery. Warren and Eberhard considered vomiting to be a highly diagnostic point and of some value prognostically, for it generally occurs rather late and is likely to indicate a lesion high in the jejunum, large in extent, and not amenable to surgery. Meyer listed vomiting as definitely occurring in 55 per cent of ninety-two cases of mesenteric

vascular occlusion. Gambee stated that in his case involving the inferior mesenteric vessels, vomiting did not occur, but claimed that vomiting is as frequent and copious as in other forms of acute intestinal obstruction, and that blood in the vomitus suggests mesenteric vascular occlusion. Mathews remarked that when hematemesis occurs the chances of recovery are poor. Boyce and McFetridge found that vomiting tends to diminish after six to eight hours when the stomach and intestines are emptied. They found peristalsis is reversed only above the lesion while in the involved area there is no peristalsis in either direction.

Bowel Function. Constipation was considered by Whittaker and Pemberton to be more common than diarrhea, and blood was noted more often in cases of venous thrombosis (37 per cent). Gambee and Warren and Eberhard, with others, considered diarrhea and melena as differential points in obstructions with and without interference to circulation. Meyer found that diarrhea occurred in 10 per cent of both the venous and arterial types. Klein designated one of his groups as the diarrheal group, with an incidence of 15 per cent. Brown considered hematemesis to be less frequent than melena and quoted Reich as stating that 41 per cent showed diarrhea and that 20 per cent showed blood in the stools. Donaldson and Stout stated that occult blood is uniformly present in the stools, and the bowel movements continue dark and soft to liquid in nature. Boyce and McFetridge considered tenesmus to be the more usual form of disturbed bowel function.

Distention. This symptom and sign is considered by most writers to be a late manifestation and is not relieved by bowel movement or enemata. Its occurrence may then be considered a grave prognostic sign.

Shock. There is unity with the opinion of Boyce and McFetridge that shock, which is marked in fulminating cases, is in other cases proportional to the degree of mesenteric involvement.

PHYSICAL FINDINGS

Temperature. The temperature at the onset is either normal or subnormal, but within a few hours may rise to 101° or 103°F.

Pulse. The pulse rate may be slow at the onset but varies with the temperature and degree of shock. In severe shock it may be rapid and thready in character.

Pallor. Those patients with a rapid onset and very severe pain will show varying degrees of pallor but the patients with venous thrombosis and a slow insidious onset may show no complexion changes until the occlusion becomes complete and the epigastric pain is severe.

Tenderness. All writers agree that tenderness is much less than expected in proportion to the pain and extent of the lesion. There is general abdominal tenderness without localization, especially in the venous cases, until the occlusion becomes complete. The tenderness in these advanced cases then tends to be epigastric.

Rigidity. This physical finding is generally lacking in the early cases, but as the lesion advances it becomes more definite as the parietal peritoneum becomes irritated. This sign may be of great differential value in eliminating other possible lesions, such as perforated ulcer of the duodenum or stomach, appendicitis, pancreatitis, and acute cholecystitis. The costal excursions may give the best index as to the presence or absence of rigidity.

Palpable Mass. Although a mass has been reported to be palpable in some cases, this cannot be depended upon for any aid in diagnosis.

Peristaltic Sounds. Those patients in whom only a portion of the bowel is involved may on auscultation show some sounds present. They are not likely to be so loud and gurgling as in cases of mechanical obstruction, but when the entire small bowel is affected by the thrombotic process, absence of peristaltic sounds is the rule, and distention is more likely to occur.

LABORATORY FINDINGS

Bloody Stools. There may be no evidence of gross blood in the stools but laboratory testing would undoubtedly show its presence more often than is reported. Some writers agree that this is a very diagnostic procedure.

Leucocytosis. At the onset the white blood count may be normal, but as the lesion progresses, leucocytosis is the rule. Whittaker and Pemberton found that the range of the blood count for venous thrombosis is 20,000 to 27,000. Polymorphonuclear predominance occurs as the count rises.

Roentgenographic Interpretation. The zero film of the abdomen could be expected to show evidence of intestinal obstruction in a large percentage of cases. It would be logical to state that the more advanced the occlusion at the time of the Roentgen examination the more clear cut the obstruction would be. Fluid levels could be expected with overlying gas.

CASE REPORTS

Case I. A white Austrian male 52 years old complained that three weeks before his hospital admission January 6, 1939, pain developed in his left upper abdomen following an alleged strain while at work on a W.P.A. project. The pain was sharp and stabbing at the onset but became constant and dull with occasional superimposed sharp stabs. He had slight hematemesis the day after the injury but denied any other gastrointestinal symptoms other than an occasional unexplained diarrhea during the past year. During the first night of hospitalization, the epigastric pain became sharp and colicky and spread throughout the abdomen. This was followed by hematemesis of bright red blood and bowel movements containing large amounts of unclotted blood. In about eight to ten hours, distention began to develop. His past medical and family histories gave no aid in the diagnosis.

Physical examination showed a white asthenic male apparently quite ill. The sclera showed a suggestion of jaundice. The lungs were clear except in the left lower lateral chest where there was percussion dullness. The heart was normal in size with normal sounds. Blood pressure was 150 systolic and 80 diastolic, while the pulse was regular at a rate of 100. There was tenderness and distention of the abdomen with limitation of epigastric excursions. Tenderness was acute in the left upper quadrant and epigastric rigidity was present. The liver and spleen were not palpable, but there was a reducible right indirect inguinal hernia. Temperature on admission was 98.4°F., but dropped to 97.2°F. after the onset of acute pain. The blood count on admission was: 90 per cent hemoglobin; R.B.C. 4,720,000; W.B.C. 9,350 with 83 per cent polymorphonuclears, 15 per cent lymphocytes, and 2 per cent eosinophiles. The urine was negative except for a trace of albumin. From these data a clinical diagnosis of acute intestinal obstruction was made.

A zero film of the abdomen showed many loops of distended small intestine arranged in the herringbone pattern and presenting various fluid levels. Pneumoperitoneum was not present.

A laparotomy was performed showing hemorrhagic infarction of the entire small bowel, except for 6 inches of the jejunum and 10 to 12 inches of the distal ileum, and the peritoneal cavity contained large amounts of serosanguineous fluid. The spleen was enlarged to six times normal size, while the liver was more firm than normal to palpation. The mesentery throughout was edematous, pale, and contained hard palpable nodules. An enterostomy was performed in the lower ileum and the abdomen was closed.

Death occurred about four hours after operation and autopsy showed marked cirrhosis of the liver, Banti's type splenomegaly (on microscopic section), extensive thrombosis of the superior mesenteric vein and its branches, and hemorrhagic infarction of nearly all of the small bowel. The mesentery was thickened, nodular, and immobile. The superior mesenteric artery, and splenic and portal veins were free of thrombi. There was no endocarditis nor arteriosclerosis.

This case represents a typical venous occlusion with the usual slowly progressing onset climaxed by severe pain associated with nausea, vomiting, bloody diarrhea, and characteristic laboratory and roentgenologic findings. This case supports the statistics from the study of 104 cases.

Case II. A female, aged 57 years, was admitted to the Robert Packer Hospital on January 2, 1937, with a history that, on the

previous day, after a long automobile trip, she began to have a colicky pain throughout her abdomen. The pain continued during the night and she vomited repeatedly. On the day after her illness began, the pain localized to the right lower abdomen and the vomiting continued despite the absence of distention. Her bowels moved normally. This patient had no previous similar attacks and her family history was irrelevant.

A physical examination showed the patient to be in some distress, temperature 98.4°F. and heart rate 96 with regular rhythm. The blood pressure was 144 systolic and 86 diastolic. The abdomen was tender in the right lower quadrant, with some rigidity but no distention.

Laboratory studies showed the hemoglobin to be 84 per cent; R.B.C. 4,000,000, W.B.C. 9600, with 96 per cent polymorphonuclears and 4 per cent lymphocytes.

An appendectomy for acute appendicitis was performed approximately twenty-six hours after the onset of the illness. The peritoneal cavity contained excessive amounts of turbid fluid. Further exploration showed 72 inches of lower ileum to be hemorrhagic, and the involved segment was resected with repair by lateral anastomosis. Her postoperative course was stormy with temperature ranging between 101.8°F. to 105°F. and the pulse rate varied between 130 and 150. Oliguria developed on the fifth postoperative day despite the daily administration of saline and glucose, and death occurred on the seventh postoperative day.

The autopsy report confirmed the surgical findings of venous occlusion near the base of the mesentery but no extension had occurred postoperatively. The enterostomy and anastomosis had not leaked.

To the author, this case represents a typical mesenteric venous occlusion with a relatively short period of onset. The etiologic factors concerned were probably the acute appendicitis and the age of the patient.

Case III. A male, aged 67 years, came to the Robert Packer Hospital on December 12, 1938, complaining that he had been awakened from his sleep by epigastric pain. Immediately after breakfast his pain became more severe but did not radiate, and he vomited twice. After a hypodermic of morphine, he was referred to the hospital. His past medical and family histories were irrelevant.

Physical examination showed the pulse rate to be 72 and his admission temperature was 96.6°F. Tenderness with slight rigidity was present over the McBurney area but there were no masses or distention. The heart was enlarged to the left with a diastolic murmur at the apex and aortic areas. Blood pressure was 210 systolic and 140 diastolic. An easily reducible indirect inguinal hernia was present on the left side.

Laboratory studies showed the urine to have only a faint trace of albumin; W.B.C. 10,000 with 88 per cent polymorphonuclears and 12 per cent lymphocytes. Two and one-half hours after admission, the leucocyte count was 18,200, with 97 per cent polymorphonuclears and 3 per cent lymphocytes. A clinical diagnosis of acute appendicitis was made and operation was advised but the patient refused surgery. Five hours later the white blood count reached 20,900 and the differential count was 92 per cent polymorphonuclears and 8 per cent lymphocytes. At this time the red cells numbered 5,200,000 and the hemoglobin was 103 per cent. The abdominal pain persisted with the pulse rate rising to 100 and the temperature was 97°F. Finally, the patient accepted surgical advice but during operative preparation, the pulse rate increased to 130 and was very weak. His blood pressure dropped to 80 systolic and 60 diastolic. In view of this rapid change of condition, the lesion was considered to be a coronary occlusion and not appendicitis, with consequent return of the patient to his bed without operation. The shock increased and death occurred approximately twenty-three hours after the onset of the illness.

Post-mortem examination showed that the peritoneal cavity contained a large amount of thick bloody fluid. The proximal jejunum for about 1 meter, and the distal half meter of the ileum were normal while the intervening small bowel was hemorrhagic, distended and friable. The arteries of the mesentery were patent and the veins were filled with ante-mortem clots. The heart was enlarged and showed severe coronary sclerosis. The final diagnosis was venous mesenteric occlusion.

This was a typical case with subnormal temperature, epigastric pain, vomiting, progressive blood changes and development of shock while the patient was under observation. However, the extent of involvement of the small gut precluded any chance of recovery even if the patient had accepted the surgical advice when he was first approached. The most important

point in the diagnosis of mesenteric vascular occlusion is to keep it in mind.

TREATMENT

Although enterostomy has been advocated by some surgeons as the method of treating this lesion, most writers agree that primary resection of the involved portion of the intestine is the best approach to this problem. It has been pointed out that resection 3 to 6 inches on either side the area of infarction is necessary to prevent extension of the thrombosis. In those cases due to over-indulgence in alcohol, as pointed out by Ochsner, resection may not be necessary to effect a cure. In patients in whom thrombosis results from infection such as appendicitis, Jones advocates the Wilms-Braun operation of ligating the ileocecal vein to prevent the extension of the thrombosis.

SUMMARY

About 772 cases of mesenteric vascular occlusion, including arterial and venous types, have been reported in the literature.

A statistical study of 104 cases of mesenteric venous occlusion from the literature, the Robert Packer Hospital, and from the author's experiences is presented.

Three rather typical cases of mesenteric venous thrombosis are reported.

The author wishes to express appreciation to Dr. Donald Guthrie, Chief Surgeon of the Robert Packer Hospital, Sayre, Pennsylvania, for permission to report two cases from his files and to Dr. K. W. Woodhouse, Fellow in Surgery, for his careful summaries of the cases.

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REDUCING THE MORTALITY OF PERFORATED APPENDICITIS*

A STUDY OF ONE HUNDRED CASES

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APPROXIMATELY 170,000 persons in the United States died of acute appendicitis during the years 1928 to 1938. Year after year, regardless of the great advances in medicine, surgery, and anesthesia, the death rate of this disease continues to exceed that of thirty years ago. Occasionally, as the result of a lay educational campaign, such as the one conducted in Philadelphia, a local decrease in the death rate has occurred. Yet in 1900, the death rate for 100,000 population from this disease in the United States was 9.7; in 1936, the latest figure available was 12.8.

During this period of thirty years, countless addresses have been presented before medical societies on the diagnosis and treatment, the choice of anesthesia, and the preferred surgical technique in appendicitis. Thousands of medical papers have exhaustively covered other phases of the subject, such as the indications for immediate or delayed operation, the choice of incision, or treatment of complications. Large series of cases have been reported to demonstrate the preference of one method over another. Many of the figures quoted have dealt with cases of nonperforated, acute, subacute, and chronic appendicitis.

In this era of medicine and surgery, there should be no mortality from appendicitis except in perforated cases. True, a tragedy may occasionally result from an error in administering anesthesia, an embolism, pneumonia, coronary occlusion, diabetic acidosis, renal failure, tetanus, or even peritonitis in nonperforated appendicitis, and every experienced surgeon has probably been unfortunate enough to observe a few such cases.

People die of appendicitis today just as they did thirty years ago because the appendix is allowed to rupture. Often this is the result of unfortunate delay on the part of parents in calling a doctor, delay frequently based on ignorance or fear, delay because either husband or wife fear surgery or its expense, delay because distance or transportation prevented the physician from reaching the patient in time, or because he or the consultant failed to appreciate the seriousness of the condition. Such failures are usually inexcusable, although perhaps no experienced internist or surgeon can say that he has never been wrong in his diagnosis of appendicitis. Because the diagnosis occasionally may be puzzling and since the mortality in nonperforated appendicitis is almost negligible, it would seem advisable to operate when in doubt. Time and experience alone teach the pitfalls, such as unsuspected pneumonia, pyelitis, salpingitis, and other nonsurgical conditions, which must be avoided in doubtful cases.

In any discussion of appendicitis, the results of large series of operations performed on simple acute and chronic appendicitis are of little value from a mortality standpoint. Guthrie[1] feels that to include uncomplicated cases in the computation of mortality in acute appendicitis is to obscure the real cause. This study, therefore, is based on a series of one hundred consecutive cases of perforated appendicitis observed over a period of fourteen years from January 1, 1924, to January 1,

* Statistical study prepared by Rollin Perkins, M.D.

1938. Five times this number of cases could be reviewed and perhaps more could be learned, but by studying carefully a group

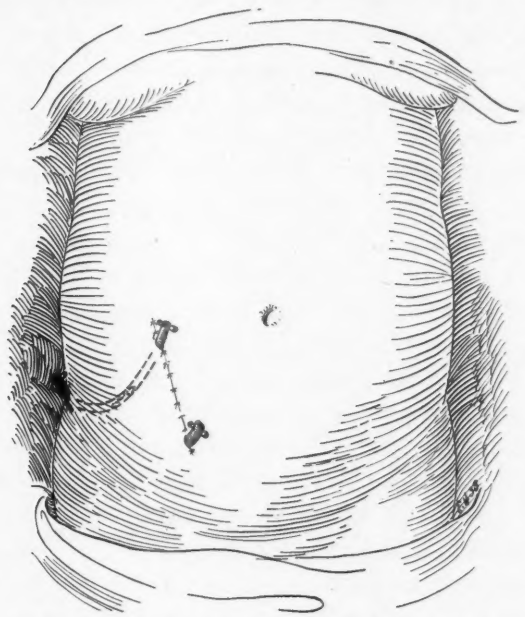

Fig. 1. Method of counter or flank drainage in perforated appendicitis with peritonitis.

this size in which the termination was fatal in twelve, many important facts about appendicitis should be revealed.

STATISTICAL STUDY

Of one hundred cases of ruptured appendix, sixty-four were male and thirty-six were female patients.

By referring to Chart 1, the average case is seen to be a male patient, 31½ years of age who had had symptoms for about three and two-thirds days before coming to see a doctor. The usual symptoms were epigastric pain or generalized abdominal pain, localizing in the right lower quadrant, associated with nausea, vomiting (usually once or twice), and probably either constipation or diarrhea. His postoperative course was a slow but uneventful recovery and he was discharged on his twenty-fourth postoperative day.

On the average, women wait longer than men before seeing their doctor. In this series, the average duration of symptoms in the female patient was 4.6 days, for men 3.3 days.

In spite of this fact, the condition is more serious in the male patient. Complications occurred in twenty-four of the sixty-four

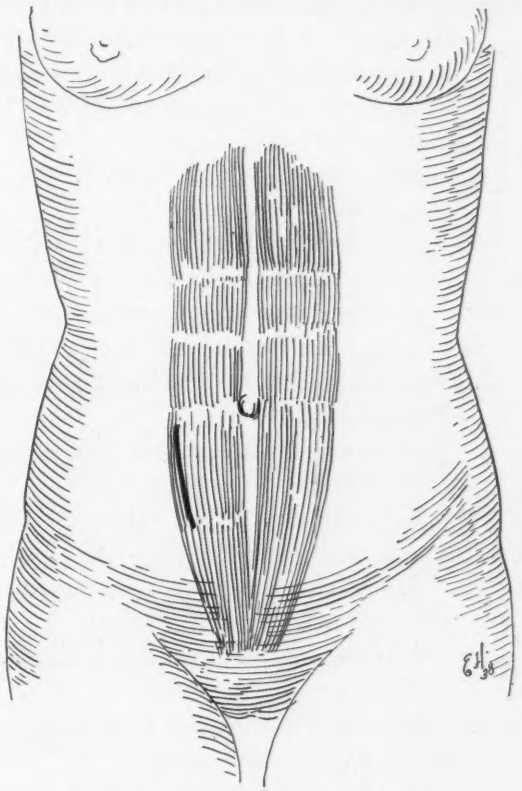

Fig. 2. Location of pararectus incision.

male patients, or in 36 per cent of the cases, but in only eight in the thirty-six female patients or 22 per cent of the cases. Furtheremore, the mortality rate was lower in the female patients, probably because of the greater resistance of the pelvic peritoneum. There were ten deaths in male patients, or 15.6 per cent, in contrast to the two deaths in female patients or 5.5 per cent. This is comparable to the death rate for the United States; in 1936, 9,989 men and 6,491 women died of appendicitis.

The mortality rate in the one hundred cases was 12 per cent.

The average stay in the hospital for patients who recovered was 24.5 days, being approximately the same for both sexes. The men were discharged on the average in 24.7 days, the women in 24.3 days.

The majority of the patients (68 per cent) recovered with no complication,

including one girl who had bronchopneumonia simultaneously with her ruptured appendix. Of the thirty-two who developed complications, there were:

1. Six cases of general peritonitis, two of which were associated with intestinal obstruction, all of which terminated fatally.
2. Four cases of fecal fistula.
   a. Two recovered.
   b. One was associated with intestinal obstruction and died.
   c. One was associated with repeated hemorrhages and died.
3. One case with hemorrhage from the wound—recovered.
4. Two cases of intestinal obstruction alone which recovered.
5. Four cases of wound infection all of which recovered.
   a. One with a chronic, suppurating abdominal wound.
   b. One with a chronically draining sinus.
6. Four cases with chest complications, all of which recovered.
   a. One developed pleurisy.
   b. One developed fluid in the chest.
   c. One developed empyema.
   d. One developed a lung abscess.
7. One case developed a subphrenic abscess and died.
8. Two cases developed thrombophlebitis, both recovered.
9. One case developed a toxemia postoperatively but recovered.
10. One case developed an abscess over the coccyx and recovered.
11. There were two postoperative hernias, both of whom recovered. One of these, a boy thirteen years of age, had a simultaneous acute tonsillitis.
12. One patient developed a pelvic abscess but recovered.
13. One developed septicemia and myocarditis and died.
14. One case, which recovered, developed abdominal distention, vomiting, and hiccup for the first five postoperative days.

In Chart II, the cases are distributed throughout the age groups, bearing out the old adage that appendicitis is a condition occurring any time from the cradle to the grave. The youngest patient was 3 years of age, the oldest 75. The average age for all patients was 31½ years. The average age for male patients was 35 years, for women 25¼.

The largest number of cases occurred in the 10 to 19 year age group, and forty-two of the one hundred cases occurred in the 3 to 19 year age groups. Furthermore, the cases in these two groups had a much better prognosis, there being 26.4 per cent complications and a mortality rate of only 9½ per cent. In the very young, the general observation is that the mortality is high, due to the greater virulence of the infection, a tendency to early perforation, and a faster spread of the pathologic alterations. Diagnostic difficulties resulting in delayed operation, and the more frequent recourse to cathartics combine to give a mortality rate of 20 per cent in children under the age of three years.[2] In elderly patients, the mortality rate increases as in other conditions because of the frequency of diagnostic errors and decreased resistance. It is interesting that of the twelve cases in the 40 to 49 year age group, there were only 25 per cent complications.

We can draw the following conclusions from this study of perforated appendicitis.

1. It is nearly twice as common in male patients.

2. It is much more serious in male patients (complications occurring in a ratio of about 3:2 as to women and the mortality rate in a ratio of about 3:1).

3. It is more common in patients from 1 to 19 years of age and the prognosis in the 10 to 20 year age group is very favorable. In the very young this is not true.

4. The choice of anesthetic makes a very marked difference in the prognosis of the case. With spinal anesthesia but two deaths occurred in forty cases; there were six

deaths in thirty-five patients when inhalation anesthesia was used.

5. Patients wait on an average of 3.6 days before seeing their physician.

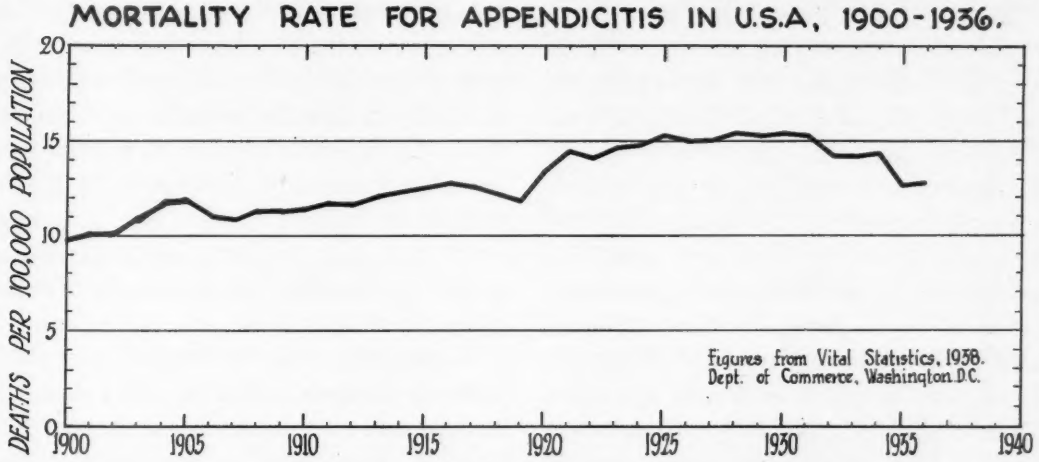

Chart I.

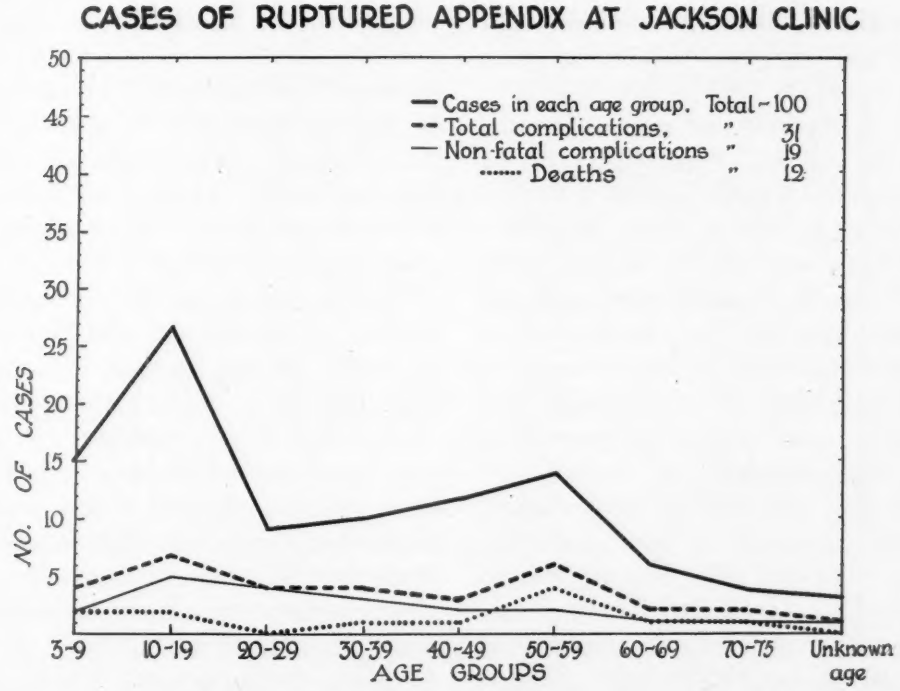

Chart II.

Garlock[3] has pointed out that the mortality rate of acute appendicitis is directly dependent upon the length of time between the onset of symptoms and operation.

FACTORS THAT WILL DECREASE MORTALITY

Eighty-one of these one hundred patients with perforated appendices and peritonitis recovered in spite of delayed surgery. Twelve lives were sacrificed to delay. All but two had taken or been given a purgative. Even the taking of castor oil or some other laxative might not have resulted fatally if operation had been performed sooner. In his Philadelphia study, Bower[4] found that in patients with appendicitis complicated by spreading peritonitis who had taken a laxative, the chance of recovery was only one in seven. Purgatives, perforation, and peritonitis lead to death in appendicitis and this fact must be impressed upon the laity if the mortality rate is to be lowered. The place to begin is in the schools.

Why was the death rate of perforated appendicitis in the United States higher in 1936 than in 1900? One explanation is that thirty years ago appendectomies were performed largely by a few skilled surgeons. Today too many appendectomies are done by physicians who have had little surgical training and inadequate postgraduate study. Interns are often deceived by the rapidity and skill with which an experienced surgeon removes an appendix, and after a year's training, they feel qualified to operate for appendicitis. Occasionally the removal of a gangrenous retrocecal appendix may be a difficult problem for even a skilled surgeon and the question of what to do in the presence of a perforation is not a decision for the inexperienced surgeon to decide. Hertzler[5] feels that the operation for acute appendicitis taxes the resources of the surgeon more than any other common lesion within the abdomen.

If the mortality rate for appendicitis is to be lowered in this country, a correct diagnosis must be arrived at in a higher percentage of cases before perforation has occurred. There is nothing new in this assertion—it has been made over and over since the late nineties. However, because a patient has a normal leucocyte count, a normal temperature, or only slight abdominal tenderness, cases of retrocecal gangrenous appendicitis are continually being overlooked and allowed to perforate. An enterolith impacted in the base of an appendix may exert such pressure on the arterial supply as to result in early, complete death of all its tissues. Cessation of pain, local tenderness, and rigidity result. Leucocytosis and other symptoms of inflammations may be lacking because, without a blood supply, there is no inflammatory reaction; there is simply gangrene as in a completely strangulated hernia. Usually there is an elevation of either the white blood count or the temperature in such cases, but failure to elicit tenderness is often typical, especially in fleshy persons. In this series of cases, the examining physician was not infrequently misled by this failure, with the unfortunate result that operation was delayed too long. A most helpful method to recognize early these deceiving cases of retrocecal appendicitis is to place the patient on his left side with his knees drawn up, have him take a deep breath, and at the end of expiration, deeply palpate at McBurney's point or higher. If the appendix is retrocecal and acutely inflamed, this pressure will elicit pain. In spite of other negative findings, our experience and judgment must be the determining factor in deciding upon the advisability of operation.

A survey by the Metropolitan Life Insurance Company showed that the highest death rate for appendicitis in this country was in the Rocky Mountain and midwestern states. The only explanation offered for this is the great distances that patients must be transported. This interesting conjecture arises: Will the building of many small town hospitals so shorten the distance of transportation, thereby lessening delay, as to offset the advantage of the larger medical centers and the more experienced surgeon?

The introduction of the routine use of spinal anesthesia at the Jackson Clinic in 1928[6] proved to be a definite factor in lowering the mortality for appendicitis. In this series of one hundred perforated appendices, seven complications occurred in thirty-three patients when nitrous oxide and ether were used, with six deaths. There were fourteen complications in forty patients when spinal anesthesia was used, with only two deaths. Many reasons could be given to show why its routine use has decreased the death rate for appendicitis, but from an experience of sixteen years, the first six of which were spent operating with inhalation, and the last ten with spinal anesthesia, the following comparison may be made: In perforated appendicitis, shock is minimized, the operation is simplified, and fatal postoperative complications are decreased under the latter. When the abdomen is opened, the intestines are quiescent and contracted under

spinal anesthesia, and the appendix may be quickly and easily located and removed with very little spreading of infection. Occasionally, when it is necessary to use inhalation anesthesia, as in some children, it seems like turning the calendar back ten years in the history of surgery. In our experience, spinal anesthesia has definitely contributed to lowering the mortality rate for appendicitis, no deaths having occurred at the Jackson Clinic from the years 1931 to 1938 inclusive.

Statistics have been quoted to show both that it is advisable to operate immediately in all cases of perforated appendicitis or that the delayed, so-called Ochsner method, is preferable. It does not seem desirable to advocate any inflexible set of rules regarding the treatment of this condition; rather each case should be considered a special problem. Undoubtedly few surgeons would advise immediate operation in a case of appendicitis perforated for three or four days with resulting spreading peritonitis, toxemia, and dehydration. Nor do many surgeons advocate the use of an ice bag and morphine when the perforation is a few hours old. It has been our experience that whenever possible it is better to operate as soon as the patient's condition permits and to remove the appendix rather than merely establish drainage. If, however, perforation is days old and either spreading peritonitis or localizing of an abscess is occurring, immediate operation is contraindicated. In such instances, the time of operation must be determined by the individual case. It may be a question of days, weeks, or even months.

A great deal has been said and written about the type of incision and how it may affect the end result of operation. Reid,[7] as well as many other surgeons, feel that there is definitely a lower mortality rate when the McBurney rather than the right rectus incision is used. Whether the McBurney, the pararectus, or right rectus incision is used has been of no great consequence in our experience since they have all been employed with the same results. When the diagnosis of acute appendicitis is apparent, and the appendix is not too difficult to remove because of abnormal location, the McBurney incision is ideal. As we may be mistaken in our diagnosis of an acute surgical abdomen and because the appendix is often not at McBurney's point, the exposure obtained by this incision is not ideal. It is not to be compared with that obtained by the pararectus incision in which the rectus muscle is withtracted toward the median line, nor have any cases of postoperative hernia resulted. I believe one of the reasons why Starr Judd was a great surgeon was because of the remarkable exposure he obtained in operating, making his operations seem simple by comparison. He showed the fallacy of small incisions and inadequate exposure in abdominal surgery. Because there is less bleeding and trauma in the pararectus incision, we have, for the past ten years, preferred this incision to the right rectus in operating for appendicitis. Where it is undesirable to break down adhesions and spread infection as in a localized abscess, the McBurney incision may be preferable.

In cases of ruptured appendicitis treated surgically, the question always arises as to the advisability of removing the appendix or merely to drain. It has been our policy to remove the appendix unless an abscess was so localized that its attempted removal might result in too much trauma and spread of infection. This was true in only three of the cases in this series. In surgery as it was practiced a quarter of a century or more ago, when the patient came to operation badly dehydrated and was then drugged with ether, the time spent on the operating table might have been a deciding factor against removal of the appendix. Today, with intravenous therapy, spinal anesthesia, and duodenal drainage in common use, time is not such an important element in the lowering of mortality.

The question of drainage is of the greatest importance in the prevention of postoperative complications and lowering

the death rate for appendicitis. Of the one hundred cases in this series, thirty were operated upon by one of us (A. S. J.) and there was only one fatality in a case of generalized peritonitis, and no serious postoperative complications. The absence of postoperative pneumonia, subphrenic or other abscesses, fecal fistula, intestinal obstruction or other sequelae may be attributed to good fortune, a small series of cases, proper drainage, and to the early anticipation and prevention of complications.

Every case with free pus in the peritoneal cavity was drained with a cigarette drain to the site of the abscess or, if there was generalized contamination, to the pelvis. In addition, counterflank drainage was used in cases of the latter type. This procedure was, I believe, largely responsible for the non-occurrence of a subdiaphragmatic abscess. Moderately soft rubber drainage tubes were used and, most important, they were not disturbed for five or six days after their insertion unless no drainage was observed by the third day, when the drain was slightly loosened. Large, fluffy, moist dressings were applied lightly so that they would not act as a cork for the drain, but encourage drainage. Gentle irrigation through the counterdrain extending from the upper third of the incision and through a stab wound in the flank was begun on the third day. Small holes cut in the middle of this tube permitted pus to be washed out with Dakin's and saline, preventing an accumulation in this trough. The cigarette drain was gradually shortened for four or five days and then replaced by a small soft rubber tube which, in turn, was gradually removed. After seven to ten days a smaller caliber drain was sutured to the end of the counterdrain and the latter withdrawn, thus drawing the new tube in place. When this was ready for removal, a piece of chromic catgut was fastened to this tube which was in turn withdrawn. This permitted gradual closing of the infected drainage tract without abrupt interruption and abscess formation.

The question of drainage has been discussed in detail because it is perhaps the most neglected subject in the literature and still the most important to the younger surgeon. We have had no experience in the closure of peritonitis cases without drainage. Improper drainage and too early removal of drains is a definite factor in the high mortality of this condition and to my brothers, Reginald and James, I wish to acknowledge advice and assistance especially in this respect.

There are few operative procedures in which the judgment acquired only by means of experience means more to the younger surgeon than in the care of cases of perforated appendicitis. The matter of wound closure is one that may easily confuse the novice. Gamble[8] was able to reduce his mortality for peritonitis due to appendicitis to 1.5 plus per cent by leaving the abdominal wound open to the air. He lost only two patients in a series of 129 cases, certainly a splendid record. I have not used this method and in fact have closed all wounds, although not tightly, with interrupted ligatures, leaving a free drainage space around the tubes. This method has so far proved successful and no postoperative hernias have resulted.

Early in my training, I became convinced that most postoperative complications were preventable; this was clearly shown to me by Dr. William J. Mayo, when I saw him operate in the most difficult cases month after month without the loss of a patient. More than any surgeon I have ever known, Dr. Mayo was able to anticipate complications and prevent them. He did not care so much whether we carried a stethoscope, but he always wanted a stomach tube on hand and the stomach kept empty. Dr. Judd always insisted upon withholding fluids by mouth for twenty-four to seventy-two hours or longer after an abdominal operation. The immediate adoption of these procedures did more to eliminate postoperative complications than

any other factor in my abdominal surgical cases. The advent of the Levine duodenal tube for drainage was a further step in the elimination of acute postoperative gastric dilatation, distention, ileus, and intestinal obstruction.

From Dr. Robert Coffee, I learned the importance of not allowing a patient to lie flat on his back after operation. Today we know that if a patient is so neglected he is predisposed to develop atelectasis and other pulmonary complications as well as an embolism. A properly trained intern and nursing staff can largely eliminate these complications. Twenty years ago, Dr. George Crile impressed upon me the value of digitalizing surgical patients before complications, such as venous stasis and embolism, occurred. I believe this, together with deep breathing and passive exercise and permitting the patient to be out of bed as early as possible, have accounted for the negligible occurrence of embolism in our abdominal and pelvic surgical cases.

We try to anticipate and prevent postoperative distention not only by walling off the appendix area from the rest of the abdominal cavity with moist sponges, but by handling the intestines as gently and as little as possible. Free pus is aspirated but no attempt is made to lavage the abdomen. The use of the heat cradle, duodenal suction, hypodermoclysis, and spinal anesthesia minimize distention. Since 1928, I have used morphine only for colic or an equivalent pain. In my opinion, it is contraindicated in almost all surgical conditions except for relief of pain so severe that it cannot be controlled by sedatives.

When distention became severe in two of my cases and ileostomy appeared imperative, hypertonic salt solution was used with complete success. In a 6 year old child who developed marked distention with apparent ileus on the twelfth day, 100 c.c. of a 10 per cent hypertonic salt solution was used with excellent results.

There were no deaths from postoperative pneumonia in this group of cases which is a convincing argument for the anesthetist and the anesthetic. Spinal anesthesia has definitely reduced fatal pulmonary complications in our experience. The use of carbon dioxide inhalations and posture change have been contributing factors. Such apparent minor details as the use of a warm dry gown, the avoidance of chilling on leaving the operating table, the use of moist, warm air in the room, and oxygen whenever indicated have helped to prevent chest lesions.

Through recent studies of Boothby and others, it appears that oxygen, when used in 100 per cent concentrations, may play an important part in lowering the mortality for ruptured appendicitis. It has already been used effectively in some cases of ileus.

SUMMARY

In summary, it is regrettable but none the less true that in spite of all advances in medical science, the death rate for appendicitis, as apparent in the chart, is higher in the United States today than it was thirty years ago.

During this period, the number of deaths from appendicitis in this country alone has approximated the number of persons now residing in the city of Milwaukee.

The unfortunate feature of this is that, barring rare complications, the death rate from nonperforated appendicitis is almost negligible, while the death rate for perforated appendicitis may be as high as 50 per cent in series of cases.

The greatest cause of the high death rate for appendicitis is delay on the part of someone who allows the appendix to rupture. The use of purgatives and laxatives is a predisposing cause and the inexperience of some surgeons a contributing factor in maintaining this high death rate.

Even if there is delay and perforation does occur, the mortality rate can still be lower if certain complications are anticipated and properly prevented. Adequate drainage maintained sufficiently long is important.

The use of spinal anesthesia, hypodermoclysis, duodenal suction, carbon dioxide inhalations, oxygen, postural change, digitalis, and other drugs as indicated are all helpful in the prevention and treatment of complications.

In this study, a series of one hundred cases of perforated appendicitis with localized and generalized peritonitis is reported. There were twelve deaths, a mortality of 12 per cent. It is reasonable to conclude that all of these deaths might have been prevented. During a period of fourteen years in which these cases were observed, 2,215 appendectomies on cases of nonperforated appendicitis were done without a death occurring that might be directly attributable to appendicitis.

That spinal anesthesia was a definite factor in lowering the mortality is shown by the fact that of the forty cases operated upon in which fourteen complications of various kinds occurred, there were only two deaths; of thirty-three cases operated upon under gas and ether, there were seven complications and six deaths. Under local anesthesia alone the death rate was high also, but these patients were in extremely bad condition.

If the mortality rate is to be lowered, there must be a greater distribution of facts regarding appendicitis to the lay public. There must be a wider dissemination of knowledge by postgraduate teaching regarding surgical principles in the treatment of perforated appendicitis to those surgeons who, either through training or practice, have had an opportunity to treat but few cases.

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SPONTANEOUS REDUCTION OF ACUTE INTUSSUSCEPTION IN CHILDREN*

ITS INCIDENCE AND SIGNIFICANCE IN THE DIAGNOSIS AND TREATMENT OF RECURRENT INTUSSUSCEPTION

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ACUTE intussusception which reduces itself spontaneously, though considered an unusually rare event, is probably more frequent than is generally realized. Because it may become a source of confusion in the diagnosis and treatment of obscure abdominal symptoms in children it is of practical importance.

Definitions. Acute intussusception is a well defined clinical and pathologic entity; because it nearly always leads to rapid necrosis of the bowel unless relieved, the entire fatal course of the disease is usually but a matter of days. When a child is ill for weeks or months and is then found to be harboring an intussusception, many authors have spoken of the disease as chronic intussusception. Under such conditions it is hard to believe that the lesion has been present continuously during all the time; a *permanent* intussusception can certainly not be compatible with life for more than a week from the extremely rare instances when the gangrenous gut is sloughed off into the lumen and passed per rectum. Two explanations for chronicity are possible: (1) that the lesion has been partial and (2) that it has been intermittent; only the latter group will be considered in this report. Obviously, however, if the lesion is intermittent the term recurrent intussusception should be used, and such a term, of course, implies repeated attacks with spontaneous reduction of the invagination after each attack. A similar confusion in terminology is often encountered in appendiceal disease; the term chronic appendicitis is frequently misused when it is applied to a patient with an obvious history of several previous attacks of acute appendicitis which subsided. It is more difficult in infants to obtain a history diagnostic of recurrent intussusception. However, in several cases herein reported such a history was elicited; moreover, in a few the tumor was clearly observed by palpation to appear and disappear. In the case reports in the literature, unfortunately, such a clear cut history or physical findings have not been generally described.

There is a further source of confusion in terminology which should be mentioned; the term recurrent intussusception, especially in the surgical literature, is often confined to instances recurring after operation. Thus, Thorndike,[1] in a paper entitled "Acute Recurrent Intussusception," refers to it only as a postoperative complication. Ladd and Gross[2] in a later paper also restrict this term to patients previously operated upon for intussusception. They state that ninety-three such cases have been reported in the literature; seven cases of their own are described, an incidence of 1.8 per cent. These authors use the term subacute and chronic intussusception when the duration of symptoms is over five days; they mention twenty such cases but do not describe the clinical features in detail, so that it is difficult to tell whether or not they presented intermittent manifestations of a recurrent lesion. Postoperative recurrence is not considered in this paper.

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Incidence. Surgeons occasionally perform a laparotomy in a child with a diagnosis of acute intussusception based upon all the characteristic manifestations including the palpation of a tumor, but find no such lesion. Careful exploration of the peritoneal cavity reveals an edema of the terminal ileum and cecum and a few enlarged ileocecal lymph nodes as the only evidence that the invagination had been present. Less puzzling to the surgeon but perhaps almost as discomfiting, is the spontaneous reduction of the lesion under his eyes just as the mass is delivered into the abdominal incision. These examples, however, while interesting, are not only rare but really have no significance in the clinical diagnosis of obscure abdominal disease in children.

At the bedside it is difficult to tell accurately how often recurrent intussusception really occurs. Stallman[3] who has reported the largest series of cases, observed nine among 117 children with intussusception, an incidence of 8 per cent. This author also discussed the terminology at some length and criticized adversely the use of the term "chronic" in these cases. Ladd and Gross[2] mention twenty cases as "subacute and chronic intussusception" among 372 cases, an incidence of 5½ per cent. Birkenfeld[4] reported 160 cases out of the literature, 109 of them in patients over 2 years of age. Other reports are summarized in Table 1. As indicated above, however, it is difficult to tell whether all of these cases were true examples of intermittent intussusception. In the experience of the present authors, five patients were observed during the past four years, a period during which twenty-seven cases of acute intussusception were admitted to the St. Louis Children's Hospital; this remarkably high incidence was responsible for making this report.

CASE REPORTS

Case 1. E. M., a 4 year old white female, had had many episodes apparently due to abdominal pain but had never vomited or become ill. However, she was constipated and failed to gain in weight during an entire year. Twenty-four hours before admission she again complained of slight abdominal pain but examination was negative; the mother stated, however, that blood in the stool was observed following an enema. Eighteen hours later the pain recurred and this time a mass was felt. On admission to the hospital the child was much more comfortable and the mass, at first palpable, disappeared under the examiner's hand. A barium enema went around to the cecum which was easily visualized.

The patient slept well, but the next morning again complained of abdominal pain, and the mass again became palpable. Stools showed no blood; barium enema now visualized an intussusception at about the hepatic flexure. Operation was immediately done and the mass found to involve only the distal ileum. It was easily reduced, and the appendix removed. Recovery was uneventful; there was no transient hyperpyrexia which frequently follows reduction of strangulated intestine. Since leaving the hospital the patient immediately began to gain weight and has been well since (four years).

Case 11. V. F., a 6 year old white female, one week before admission was suddenly seized with cramping abdominal pain which passed off in a few minutes. On each of the next few days similar attacks occurred but only once did she vomit. Stools were normal and contained no blood or mucus. She was kept in bed, however, and the attacks grew less severe. On the morning of admission an especially severe attack awakened her and she vomited for the second time and soon after passed blood per rectum. On her admission to the hospital a mass was felt and at operation an intussusception was found involving the cecum and ascending colon; it was easily reduced and the appendix removed. Recovery was uneventful; there was no postoperative hyperpyrexia. No further attacks occurred (three years).

Case 111. J. R., a white male infant, at three months of age developed occasional bouts of "colic" for one week, associated with normal stools. The day before admission he refused feedings and passed bloody fluid following an enema. He vomited three times. A mass was felt but disappeared a few hours later. No operation was carried out and the patient remained well for a year except for constipa-

tion; he then developed a recurrence of severe abdominal cramping, this time associated with numerous loose stools but no blood. No mass was noted and the infant recovered after two days, and has remained well (two years).

Case IV. P. J. R., a 23 month old white female, was admitted because she had vomited persistently for twelve hours, had severe abdominal pain and presented a large ileocecal mass on abdominal palpation. On her admission to the hospital the presence of the mass was confirmed by one observer, but later was definitely gone. An enema was returned with blood-streaked mucus. No further vomiting or abdominal pain occurred and the patient was discharged in two days. She has been well since, with no recurrence of bowel disturbance (two years).

Case V. E. R., a 19 month old white female, listless and ailing for two weeks, was given a "round of calomel" followed by castor oil, resulting in several loose stools. For a week the baby seemed well except for frequent desire to pass stool. One week before admission she began to have short attacks of restlessness and screaming, drawing her legs up into the air, vomiting occasionally. Various home remedies were given, including further cathartics, but the attacks became more frequent. The day of admission a physician was called who felt a mass in the abdomen, considered it to be a tumor of the right kidney and sent the patient into the hospital.

On admission an abdominal mass was felt but it diminished definitely in size under the palpator's hand. Rectal examination was negative and three large semi-liquid stools were passed without blood. The patient was dehydrated and was given parenteral fluids; she fell asleep and by the next morning the mass was gone. Several loose stools were passed during the day, none exhibiting blood. By evening, i.e., twenty-four hours after admission, the mass became definitely palpable again and operation was carried out. An intussusception was found involving the terminal ileum and proximal colon; it was easily reduced and the appendix removed. The postoperative course was uneventful except for a transient hyperpyrexia. She has remained well up to the present time (two years).

Discussion of Present Findings. Besides the five cases described above, thirty cases were collected from the literature although they obviously represent only a part of the many reported. They serve, however, to indicate the main clinical features which are tabulated in Table 1. Of especial interest is the frequency of diarrhea; ordinarily this symptom serves to detract from a diagnosis of intestinal obstruction. Yet, by the occurrence of a repeated invagination it is easy to see how an edema of the intestinal wall could occur with the production of an irritative inflammation resulting in diarrhea. In the five cases reported above, however, diarrhea was present in only two. It should also be noted that blood in the stool, while present in most cases, was absent in over one-third of them. Of interest is the fact that not all cases gave a clear history of intermittent attacks of abdominal pain; vague manifestations such as listlessness, constipation, frequent desire to pass stool, failure to gain weight, and anorexia were often noted.

Of therapeutic importance is the fact that operation resulted in cure of those patients that survived. That a manual reduction at laparotomy should tend to prevent recurrence is possibly explained by the serosal trauma resulting in the laying down of adhesions. If this is true it is undoubtedly more advantageous also to perform an appendectomy when the patient's condition permits, certainly at least in those lesions involving the ileocecal valve. Of the five cases described above, appendectomy was performed in each of the three operated on. In the remaining two cases, operation was not carried out because the attacks of intussusception rapidly subsided. Operation would seem the safest procedure, however, unless the clinical manifestations, including the palpation of the mass, definitely disappear within an hour or two. In many patients, on the other hand, the physician may always examine the child after the attack is over, fail to find a mass and thus remain in the dark as to the diagnosis. The problem of therapy in such a case is especially difficult, particularly in regard to the question of

TABLE I

SUMMARY OF CASES FROM THE LITERATURE

Author	Age (Years)	Duration of Symptoms (Weeks)	Mass	Blood in Stool	Diarrhea	Operation	Result	Remarks
Mayo[5]	3½	5	o	o	o	Resection	Well	Distention and generalized tenderness.
Epstein[6]	2	12	+	+	?	Reduction	Well	*X*-ray showed obstruction.
Alexander[7]	4	2	+	+	o	Reduction and appendectomy	Well	Diagnosis made only on palpation of mass.
Still[8]	1	6	+	o	o	Reduction	Well	Diagnosed tuberculous peritonitis.
Still[8]	2	4	+	+	+	Reduction	Well	Diagnosed tuberculous peritonitis.
Still[8]	1	4	+	+	o	Reduction	Well	Diagnosed tuberculous peritonitis.
Still[8]	3	3	+	+	o	Reduction	Well	Diagnosed tuberculous peritonitis.
Spencer[9]	7	16	+	?	+	Laparotomy	Died after operation	Treated as tuberculous peritonitis.
Owen[10]	6	3	+	o	o	Reduction	Well	Mass noted only before operation.
Marsh[11]	2	10	o	+	o	Ileocolostomy	Well	
Marsh[11]	3	10	o	o	o	Reduction and appendectomy	Well	
Beaven[12]	3	4	o	o	o	Reduction	Well	Meckel's diverticulum.
Beaven[12]	5	2	o	o	o	Reduction	Well	
Donovan[13]	1	8	+	+	o	Reduction	Well	
Salley[14]	1	4	+	+	+	Reduction	Well	
Sutherland[15]	3	8	+	o	o	Reduction and appendectomy	Well	Diagnosed tuberculous peritonitis.
Henderson[16]	1	7	o	+	+	Reduction	Well	Operation at third attack.
Stallman[3]	¾	8			o	Reduction	Well	
Stallman[3]	11	12	+	+	+	Reduction	Recurrence	Reoperation for colopexy.
Stallman[3]	5	9	+	+	+	Reduction and appendectomy	Well	
Stallman[3]	7	12	+	o	+	None	Died	Diagnosed tuberculosis. Autopsy: perforation of intussusception with general peritonitis.
Stallman[3]	7	13	+	+	+	Laparatomy	Died	Considered tuberculosis. Autopsy: perforation of intussusception with general peritonitis.
Stallman[3]	1½	5	+	+	+	Reduction	Well	
Stallman[3]	¾	3	?	+	+	Resection	Died	Two hours after operation.
Stallman[3]	8	2	?	o	+	Reduction	Well	Died in four months of general peritonitis.
Stallman[3]	2	3	+	+	+	Reduction	Well	
Jones[17]	¾	4	o	+	+	Ileocolostomy	Died	Few hours after operation.
Jones[17]	1	1	+	+	o	Reduction	Well	
Jones[17]	9	2	+	o	o	Reduction	Well	
Jones[17]	5	6		o	o	Reduction	Well	
Case I	4	?	+	o	o	Reduction and appendectomy	Well	Rapid weight gain after operation.
Case II	6	1	+	+	o	Reduction and appendectomy	Well	
Case III	¼	1	+	+	+	None	Well	
Case IV	2	1 day	+	+	o	None	Well	
Case V	2	2	+	o	+	Reduction and appendectomy	Well	

operation. Careful abdominal examination *during* the attack should, however, in nearly all cases reveal the true nature of the lesion. If no mass is present, unless the attack has been recent exploratory laparotomy may reveal little; it should be emphasized, however, that a mobile cecum with a long persistent mesocolon is a suggestive finding. Waugh,[18] in 1911, called attention to this defect in rotation as a probable etiologic factor in intussusception. The present authors have always noted such a finding in these cases. If operation is carried out, appendectomy, as already mentioned, seems definitely an advantage, provided the patient is in good condition. It is of interest to note that in none of the postoperative recurrences described by Thorndike[1] was this procedure done.

SUMMARY

Five cases of acute recurrent intussusception in children are described in which spontaneous reduction occurred after one or more attacks. In three patients operation was carried out, the intussusception reduced and the appendix removed; in two no operation was performed. Clinical cure was observed in all cases with no further attacks for a significant interval.

Analysis of these cases, as well as of thirty others culled from the literature, emphasizes the importance of recurrent intussusception as a cause of obscure abdominal complaints in children. The clinical manifestations, terminology, diagnosis and treatment have been briefly discussed.

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THE POSTOPERATIVE CONCENTRATION OF BILE SALTS IN HUMAN BILE*

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A WIDE range of functional investigations has been undertaken in an attempt to determine the degree of functional hepatic damage present in a given patient, but of all the functions ascribed to the liver only one has been proved to be exclusively and specifically characteristic of that organ. This is the synthesis and secretion of the soluble salts of the various bile acids (Foster, Hooper, and Whipple[1]).

Experimental studies of the secretion of these bile salts have indicated that a multiplicity of factors is involved in determining their concentration in the bile secreted by an individual liver. These factors have been studied extensively in experimental animals by Stadelmann,[2] Whipple,[3] McMaster, Broun and Rous,[4] Rous,[5] Greene and Snell,[6] McGowan, Bollman and Mann,[7] Doubilet,[8] and many others. Similar studies have recently been made on human fistula cases in this country by Greene, Walters and Fredrickson,[9] Sterner, Bartle and Lyon,[10] Ravdin, Johnston, Riegel and Wright,[11] Breusch and Johnson,[12] Kohlstaedt and Helmer,[13] Doubilet,[14] Gray, Butsch and McGowan,[15] and by Gray, McGowan, Nettrour, and Bollman.[16] These investigators dealt with the secretion of bile salts and acids by the liver in patients following the insertion of a T-tube and drainage of the common bile duct. The conclusions shared in common by most of these investigators will be discussed in the consideration of the results in this study.

We wish to report our experiences in the determination of the concentration of bile salts in fistula cases and to analyze the effect on the concentration of bile salts in the bile of (a) liver disease; (b) infection, including cholangitis and fever of any origin; (c) anesthesia and operative trauma; (d) dextrose administration; (e) bile refeeding and the administration of bile salts; and (f) the concentrating ability of the common duct itself.

Daily determinations of the concentration of bile salts and cholesterol and of the pancreatic ferments were performed on the bile of forty-seven consecutive patients who had operative biliary fistulae. All bile was secured by means of T-tube drainage of the common bile duct. Microscopic examination of the biliary sediment was routinely performed. The bile was cultured where an indication was present. All the tests were performed on operative bile specimens secured by aspirating the common bile duct before it was opened. Hourly determinations of the concentration of bile salts were frequently performed to determine variations in the concentration of bile salts through the day and following specific therapeutic measures. The bile salts were determined by the Aldrich-Bledsoe method,[17] the cholesterol by the Lieboff method,[18] and the ferments by the methods described by Myers and Fine.[19]

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We are not reporting the total volume of bile and therefore the total output of bile salts, for a total biliary fistula seldom occurs following the insertion of a T-tube. Such data at best would represent only an approximation.

Hepatic Damage. A decrease in the capacity of the liver to secrete and concentrate bile salts apparently parallels the degree of hepatic damage (Bollman and Mann[20]). The publications of Greene, Walters and Fredrickson, Ravdin, Johnston, Riegel and Wright, and of Gray, McGowan, Nettrour, and Bollman substantiate this observation. Ravdin and his associates found that the bile salts invariably were absent in bile following complete biliary obstruction for a week or more and that a considerable time was necessary for their reappearance after the relief of obstruction. He also reported a parallel between the concentration of bile salts in fistula bile and the degree of hepatic damage present at the time of operation. Gray, Butsch, and McGowan also studied the effect of anesthesia, various operative procedures, and postoperative treatment on the concentration of bile salts and found that the latter was inversely proportionate to the degree of hepatic damage.

We have attempted a similar study based upon the degree of hepatic damage as determined both by the preoperative clinical condition of the patient and by the appearance of the liver at the time of operation. In Table 1 the patients have been grouped according to this evaluation of the degree of hepatic damage and this factor has been compared to the concentration of the bile salts found in the common duct at operation, the rapidity of the postoperative return of the bile salts in the bile to a concentration within the lower limits of the normal range,* the proportion of subjects in which there was not a return to a low normal concentration of bile salts during the period of drainage, and the concentration of cholesterol in the drainage bile. Table 1 confirms the observations of Gray, McGowan, Nettrour and Bollman.

* 800 mg. per cent or more bile salts.

When, however, individual cases are studied in detail, no such clear cut parallelism exists, and certainly, in any given patient the concentration of the bile salts and the rapidity of their return to a normal concentration does not necessarily correspond to the degree of liver damage, nor does it indicate the functional state of that liver. An example of this is seen in the two patients (Table 1) who by all tests and by observation had a normal or nearly normal hepatic function, yet in whom at no time during the period of drainage did the concentration of bile salts approach the normal. The convalescence of these patients was so uneventful and the follow-up period so satisfactory that we do not doubt but that they have normally functioning livers. The many other factors that enter into the concentration of bile salts in fistula bile will be discussed later.

Table 1

Relation of Liver Disease to Concentration of Bile Salts and Cholesterol in Common Duct Drainage Bile

Hepatic Damage*	Cases	Average Operative C.D. Bile Salt Concentration, Mg. Per Cent	Day Bile Salts Returned to 800 Mg. Per Cent, Days	Number of Cases Not Returning to Normal	Average Daily Cholesterol Concentration, Mg. Per Cent
None.....	11	1600	5	2	50
Mild.....	14	1200	5	2	62
Moderate....	18	570	8	8	50
Severe.....	4	Trace in 2		3 (1 on twelfth day)	30 (2 had none at any time)

* As determined by history, function tests and by operative findings.

In general those patients with severe hepatic damage, especially those with complete extrahepatic obstruction of the biliary tract had no bile salts or only a very low concentration thereof in operative bile specimens. Contrary to the findings of Ravdin, Johnston, Riegel, and Wright these livers secreted bile salts shortly after removal of the obstruction, though the ability to secrete bile salts in normal concentration was regained by only one patient during the period of drainage. These

studies on the influence of hepatic damage upon the concentration of bile salts do not lend themselves to statistical analysis. We, therefore, are presenting two representative case reports from each group with a discussion of the probable factors influencing the results noted.

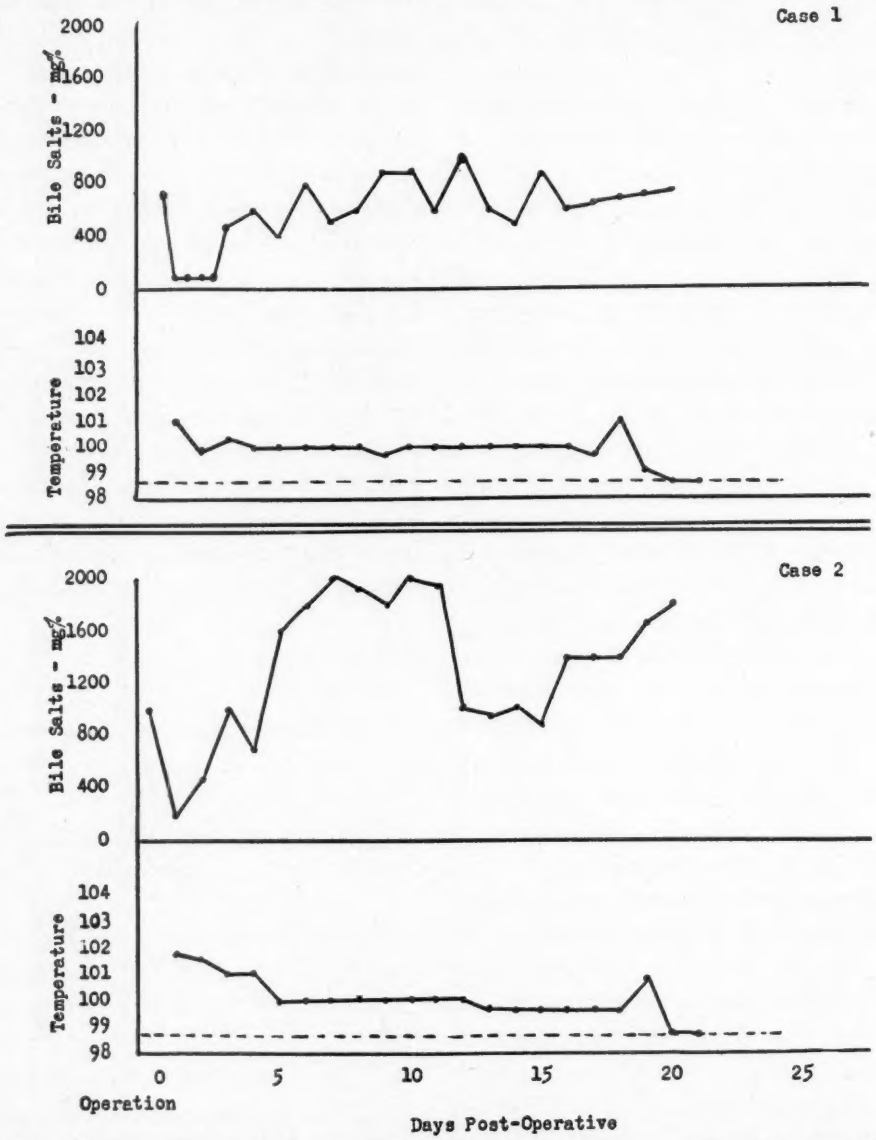

FIG. 1. The postoperative concentration of bile salts in two patients with normal livers.

THE CONCENTRATION OF BILE SALTS IN BILE FROM PATIENTS WITHOUT EVIDENT HEPATIC DAMAGE

The eleven individuals in this group cannot be considered normal, as all were operated on for disease within the biliary tract. It may therefore be assumed that they had some slight degree of hepatic involvement. These cases are, however, those in which by history and by laboratory examination, by physical examination and by appearance at operation, the liver apparently was normal. The concentration of bile salts in the bile from such livers is illustrated in Figure 1.

CASE 1. A white woman was admitted with a history of repeated biliary colic over a period of two years. Four days before admission she developed jaundice, the icteric index rising to 50 units. There was a direct Van den Bergh in the serum and a low concentration of bile salts in the duodenal drainage bile.

Upon operation the gall-bladder was chronically inflammed and contained many "mixed" gallstones. The common bile duct was dilated and contained a solitary stone much like the stones found in the gall-bladder. The cystic duct was dilated. A cholecystectomy and a choledochostomy with T-tube drainage were done. The liver was normal in appearance.

The concentration of the bile salts in the fistula bile is charted in Figure 1. The temperature curve, showing an uneventful recovery, is also included.

Case II. A white woman had a history of intermittent epigastric pain over a period of twenty years. There was no jaundice except for a mild short attack fifteen years before. On admission the blood cholesterol was 125 mg. per cent, the cholesterol esters 145 mg. per cent, the icteric index 9.0 units, and the duodenal drainage showed concentrated bile with calcium crystals and a colon bacillus on culture. The gall-bladder was not visualized roentgenographically.

Upon operation, the liver was normal in appearance, the gall-bladder chronically inflamed, containing many stones. The common duct was dilated but thin walled. It contained calcium bilirubinate stones quite different in appearance and structure from their fellows in the gall-bladder. On culture, the common duct bile contained colon bacilli though the duct was not microscopically inflamed. The concentration of bile salts and the temperature curve are shown in Figure 1.

Discussion. These two patients with apparently normal livers present the findings general to the cases we have listed as normal. In Case 1, only a low normal concentration of bile salts was attained during the twenty days of the study, while in Case II concentrated bile was secured soon after operation. In all our normal cases there was an initial postoperative depression in the concentration of bile salts which usually regained a level of 800 mg. per cent within a few days, although in several cases this was delayed up to the tenth day. Very frequently a secondary decrease in concentration occurred after six to fifteen days of drainage. This may have been due to a deficiency of bile salts resulting from the prolonged drainage through the T-tube as well as to a reduction in carbohydrate intake following the discontinuance of parenteral administration of glucose solution. Due to these and other variables, it is impossible to assess the degree of hepatic damage on the basis of the postoperative concentration of bile salts alone.

The cholesterol content of the fistula bile did not parallel the concentration of bile salts in the two cases shown or in any of the patients we studied. It was generally of a normal concentration in our normal cases, unless there was an associated cholangitis in which instance cholesterol sometimes disappeared from the drainage bile.

THE CONCENTRATION OF BILE SALTS FROM PATIENTS WITH MILD HEPATIC DAMAGE

These patients, with evidence of slight but definite hepatic disease, showed a relatively rapid postoperative return of bile salts to normal levels. The extremely variable concentrations shown in Figure 2 are due to factors to be discussed later. When the 14 cases in this group are treated as a unit, the results are consistent within the clinical classification of the amount of liver disease seen. This, however, does not hold for individual cases. We are, therefore, presenting two case reports with variable concentrations of bile salts in the fistula bile.

Case III. A 64 year old white woman was admitted with a history of epigastric pain of eighteen months' duration, associated with nausea, vomiting, and distention. Three weeks before admission, jaundice appeared. The icteric index was 15.0 units, the Van den Bergh reaction was direct. The gall-bladder was not visualized roentgenographically.

Upon operation, the gall-bladder was chronically inflamed and obliterated. The common bile duct was dilated, noncalculous, and thin walled. A chronic pancreatitis was considered the cause for the dilatation. The liver was slightly granular. A cholangiogram twelve days postoperatively showed a normally functioning common duct which emptied immediately. The concentration of bile salts in the fistula bile is given in Figure 2.

Case iv. A 39 year old woman was admitted to the medical wards with typhoid fever. At the end of the third week she was suddenly seized with severe abdominal pain and mild jaundice appeared. After three days' observation, she was operated upon for acute cholecystitis.

The gall-bladder was found to be acutely inflamed, calculous and very edematous. The liver was enlarged and discolored. The common bile duct was dilated but contained no calculi. A cholecystostomy was performed. The postoperative bile salt concentration is given in Chart. iv. In this patient the concentrating ability was definitely interferred with by the febrile reaction until after the tenth day.

Discussion. The history and the chemical work-up in these cases did not indicate any liver damage. The macroscopic appearance of the liver, however, and the evidence of severe cholecystic disease in both instances were evidence of some hepatic impairment. In both instances the liver had the ability to secrete bile salts in moderate concentration but the response was variable in both cases. This irregularity in Case iii and the delay in the return of function in Case iv apparently were not dependent on the intrinsic disease of the liver but rather on the concomitant factors of a low grade cholangitis in the one, and a

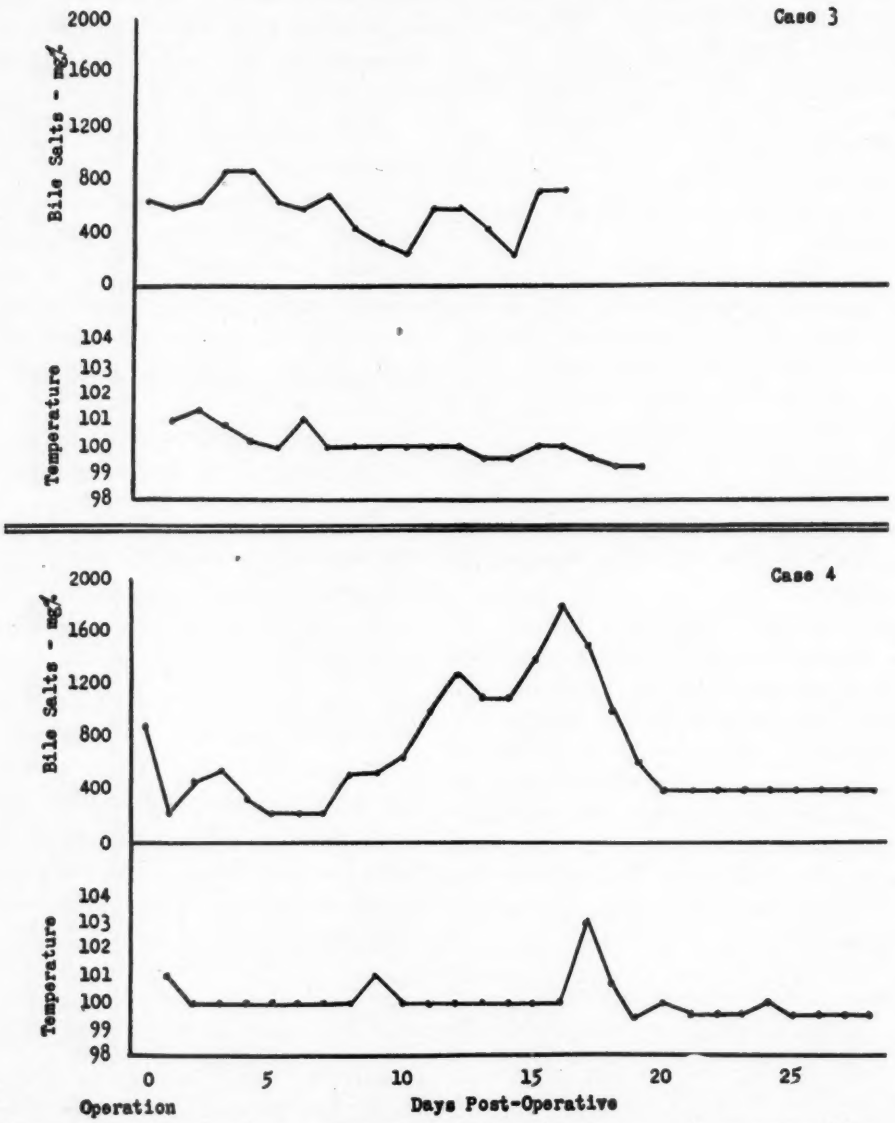

Fig. 2. The postoperative concentration of bile salts in two patients with mild or minimal hepatic damage.

general systemic infection with associated acute involvement of the gall-bladder in the other.

THE CONCENTRATION OF BILE SALTS IN PATIENTS WITH MODERATE HEPATIC DAMAGE

Prolonged partial obstruction of the common bile duct predisposes to continuing "insult" to the liver with a variable degree of hepatic damage. Eighteen such cases are included in this series. In each instance the history and the preoperative chemical findings were substantiated by the operative finding of hepatic disease. The two cases (v and vi) are representative of the functional behavior of such livers and their response to medical therapy and the removal of the obstruction.

Case v. A 61 year old female had a long history of epigastric pain radiating to the right and left upper quadrants. Eighteen months before this admission, she had had a cholecystectomy and a choledochostomy for chronic cholecystitis and choledocholithiasis. The symptoms, however, continued. Two weeks before admission she developed jaundice which was continuing in type. The blood cholesterol was 205 mg. per cent, the esters 60 mg. per cent, the icteric index 44 units, the Van den Bergh reaction direct, and the serum bilirubin 9.4 mg. per cent. Duodenal drainage showed a moderately concentrated bile containing calcium crystals and colon bacilli.

Upon operation, typical calcium bilirubinate stones were found in a dilated common duct, which was infected with colon bacilli. The liver was enlarged and granular. Chemical examination of the common duct bile showed 775 mg. per cent bile salts and a trace of cholesterol. The postoperative concentration of bile salts in this patient with liver disease and cholangitis is shown in Figure 3.

Case vi. A 57 year old female was admitted with a history of epigastric pain of eighteen months' duration. During the preceding two months she had intermittent jaundice. Three years previous to admission she had had a cholecystectomy for biliary colic from which she had been relieved. The blood cholesterol was 230 mg. per cent, the cholesterol esters 55 mg. per cent, the icteric index 170 units and the serum bilirubin 14.5 mg. per cent. On duodenal drainage the bile was dilute and contained calcium and calcium bilirubinate crystals in large amounts. A colon bacillus was found upon culture of the bile.

Upon operation, the common bile duct was markedly dilated and contained a very large calcium bilirubinate stone. The common duct bile was dilute. The liver was granular, discolored and presented evidence of a biliary cirrhosis. The postoperative chemical studies on the bile salts in the fistula bile are shown in Figure 3.

Discussion. These two patients undoubtedly had moderate to marked hepatic damage both by history and at operation. Both had secondary choledochostomies performed for recurrent common duct stones. In spite of the advanced disease, both secreted bile salts at all times even though in diminished concentration. The true degree of hepatic damage was best shown by the slow return to a moderate concentration of bile salts in the fistula bile. This at no time reached a concentration we consider as a low normal (800 mg. per cent). Case v showed a similar inability to secrete cholesterol apparently associated with the infection of the common duct by colon bacilli. This, however, was not true of Case vi which had even more severe hepatic damage as well as an active cholangitis. Both of these patients have been followed biweekly for nearly two years and show a continuing cholangitis, but they have recovered their hepatic function to a marked degree as is evidenced by a moderately concentrated bile on duodenal drainage.

THE CONCENTRATION OF BILE SALTS IN PATIENTS WITH SEVERE HEPATIC DAMAGE (COMPLETE OBSTRUCTION)

The degree and duration of an extrahepatic biliary obstruction is not necessarily an index to the degree of hepatic damage. Four subjects in our series had complete obstruction produced by stricture of the common bile duct. The response of

the liver to the removal of the obstruction was variable, but in no instance did the liver fail to secrete bile salts shortly after relief from the obstruction.

duodenal drainage, though the tube was proven to be in the duodenum.

Upon operation, there was a complete stenosis of the common bile duct. A bulbous protrusion at the hilus of the liver was identified as the dilated end of the junction of the right and left hepatic ducts. The hepatic ducts were anastomosed to the duodenum over two T-tubes. The operative bile, aspirated from the bulbous protrusion described, was white and contained no bile salts and only a trace of cholesterol. The response of this liver, which had been completely obstructed for a period of several months, is shown in Figure 4.

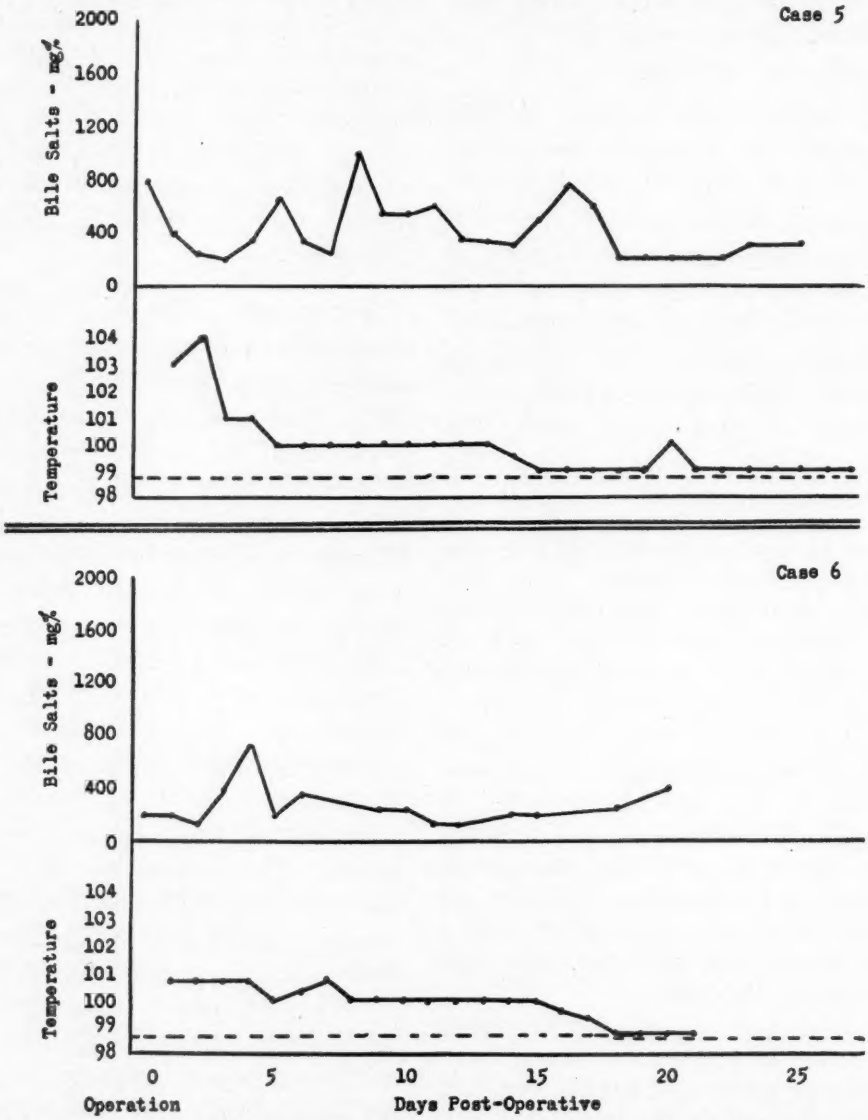

FIG. 3. The postoperative concentration of bile salts in two patients with moderate to marked hepatic damage.

CASE VII. A 42 year old woman had a cholecystectomy six months before admission to this hospital. Six days following her initial operation, she was operated on again for intra-abdominal hemorrhage. Two months after this operation jaundice appeared. It was progressive and painless though associated with nausea and vomiting. Upon admission she was deeply jaundiced. The blood cholesterol was 240 mg. per cent, the cholesterol esters 70 mg. per cent, the icteric index 186 units, the Van den Bergh direct, and the serum bilirubin 19 mg. per cent. No bile could be secured on

CASE VIII. A 58 year old woman was admitted with a history of severe epigastric pain, nausea, and vomiting of three years' duration.

These symptoms followed closely upon a cholecystectomy for chronic cholecystitis and cholelithiasis. Jaundice was severe at times of a probe after the removal of the stones and a stricture was thought to exist at or near the ampulla. A T-tube was inserted into the com-

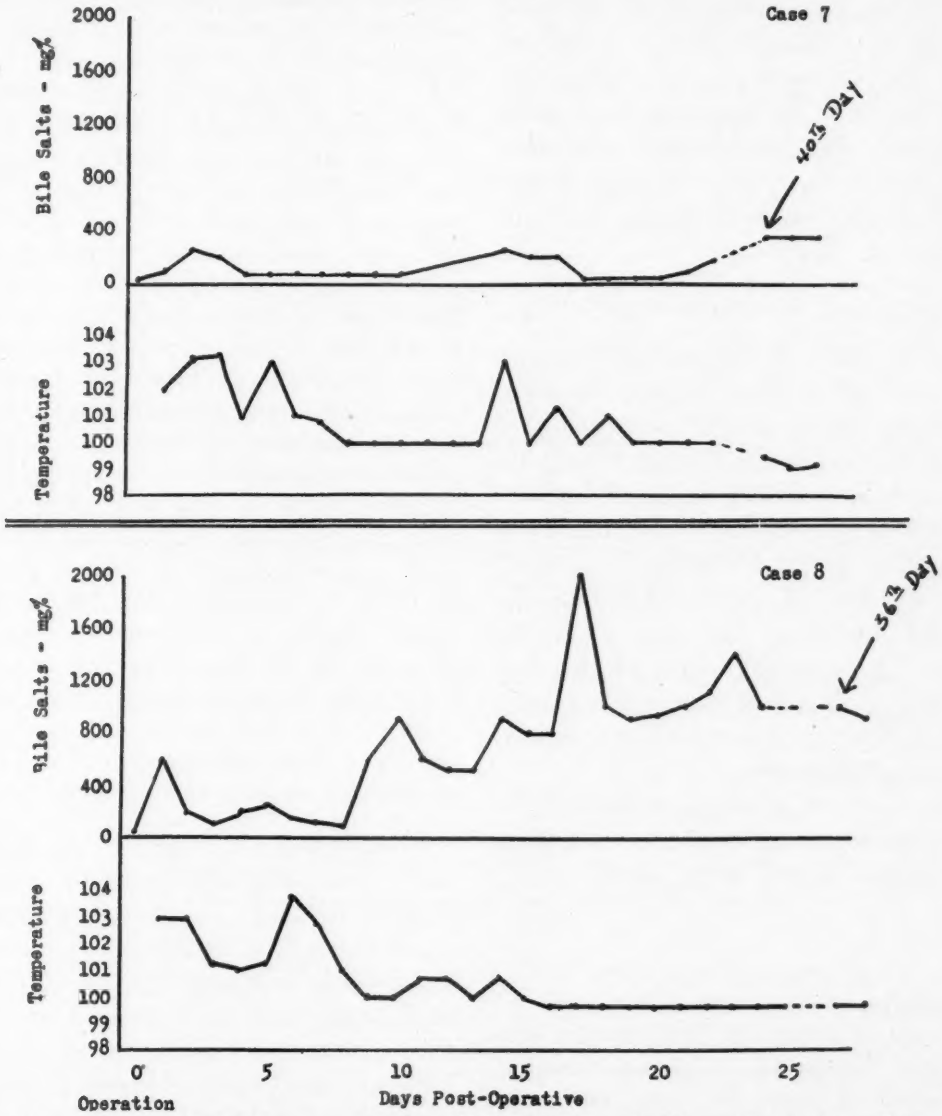

Fig. 4. The postoperative concentration of bile salts in two patients who had complete obstruction of the common bile duct with "white" bile in the hepatic ducts at the time of operation.

but apparently variable in intensity though always present. On admission the blood cholesterol was 210 mg. per cent, the icteric index 43 units, the Van den Bergh direct and the serum bilirubin only 4.3 mg. per cent. The degree of icterus increased during the patient's preoperative stay.

Upon operation, the common bile duct was found markedly dilated, containing white bile. Several pure pigment stones were found in the common duct, one of which completely obstructed the common bile duct at the ampulla. The duodenum could not be entered by means mon bile duct for temporary relief, for the liver showed advanced biliary cirrhosis. The patient, discharged on the thirty-eighth postoperative day, was still draining profusely through the common duct fistula. Unfortunately, she could not be brought back for follow-up. Figure 4 shows the response of this severely damaged liver to relief from the obstruction.

Discussion. The two cases cited were undoubted complete obstructions of the extrahepatic biliary tree. In spite of this, both responded promptly to relief from the

obstruction and bile salts appeared in the drainage bile during the first twenty-four hours. This finding is contrary to that reported by Ravdin, Johnston, Riegel and Wright who, in eighteen cases of complete obstruction of the common duct, found an absence of bile salts for seven or more days in each patient. We believe that complete absence of bile salts in the bile is of severe prognostic import and is found only in complete hepatic failure.

INFECTION (CHOLANGITIS)

Infection of the common bile duct is one of the factors other than hepatic damage that adversely affect the concentration of bile salts in fistula bile. Although it may be argued that it is a direct cause of hepatic damage, we shall consider infection as a separate entity in the present discussion. Gray, Butsch and McGowan have shown that a febrile reaction, regardless of cause, will depress the concentration of the bile salts in the bile and will retard the return to a normal concentration after the initial postoperative depression.

The two following cases represent an apparent impairment of hepatic function in patients whose livers were only mildly diseased.

Case IX. A 57 year old male had a history of mild attacks of intermittent jaundice for thirty months, with weight loss, nausea, distention, and occasional epigastric distress. Blood cholesterol was 305 mg. per cent, cholesterol esters 120 mg. per cent, icteric index 11.5 units. The Van den Bergh reaction was direct but the serum bilirubin was normal. Duodenal drainage showed a moderately concentrated bile containing calcium bilirubinate and cholesterol crystals. Cultures showed the presence of colon bacilli. The gall-bladder was not visualized roentgenologically.

Upon operation, the gall-bladder was large and edematous but without calculi. The common duct was dilated to 3 cm. in diameter and contained several calcium bilirubinate stones. On digital examination of the interior of the common duct a radial papillomatosis of the ampullary end of the duct was recognized. A cholecystogastrostomy and a T-tube drainage of the common bile duct were done. Repeated cultures of the drainage bile showed the continuing presence of colon bacilli and streptococci. The postoperative concentration of bile salts, the bile culture reports and the febrile course are shown in Figure 5.

Case X. A 48 year old woman was admitted with a history of right upper quadrant pain for the past fifteen years. There was no jaundice until three days previous to admission when it appeared, associated with dull upper abdominal pain. The jaundice increased until the patient was operated upon seven days later. The blood cholesterol was 256 mg. per cent, the icteric index 32.6 units, the Van den Bergh direct and the serum bilirubin 4.5 mg. per cent.

Upon operation, the gall-bladder was acutely inflamed and contained many calculi. The common duct was edematous, dilated but free from stones. The liver was not remarkable. The operative bile from the common duct was light in color and contained numerous pus cells and crystals. On culture, colon bacilli were identified. Cholecystectomy and choledochostomy were performed. The convalescence was stormy. On several occasions the drainage bile showed cultures of colon bacilli. Figure 5 gives the results of daily determinations of the concentration of bile salts and the febrile course.

Discussion. In neither of these cases was there any macroscopic evidence of hepatic disease. The patients were treated assiduously by means of parenteral dextrose, free drainage of the common duct and supportive measures. The concentration of bile salts from the fistulas of these patients at no time returned to the minimal normal concentration. In Case IX the determinations were continued for fifty days. Throughout this time the patient, though otherwise in good condition, ran a continuing fever with colon bacilli and streptococci cultured from the bile. The acute febrile reaction noted between the thirtieth and thirty-sixth postoperative days was induced by clamping the T-tube and so retarding drainage. The concentration of bile salts immediately fell. These findings were also present, though in a lesser degree in Case X. It would appear that a cholangitis and/or a febrile reaction will depress the concentration of bile salts

secreted by the liver. These cases show the difficulty of determining the functional state of the liver solely on the basis of the concentration of bile salts in its secretion.

nearly all the cases of this series has been ascribed by Gray, Butsch and McGowan to the effect of anesthesia. No difference was seen by us in the effects of various

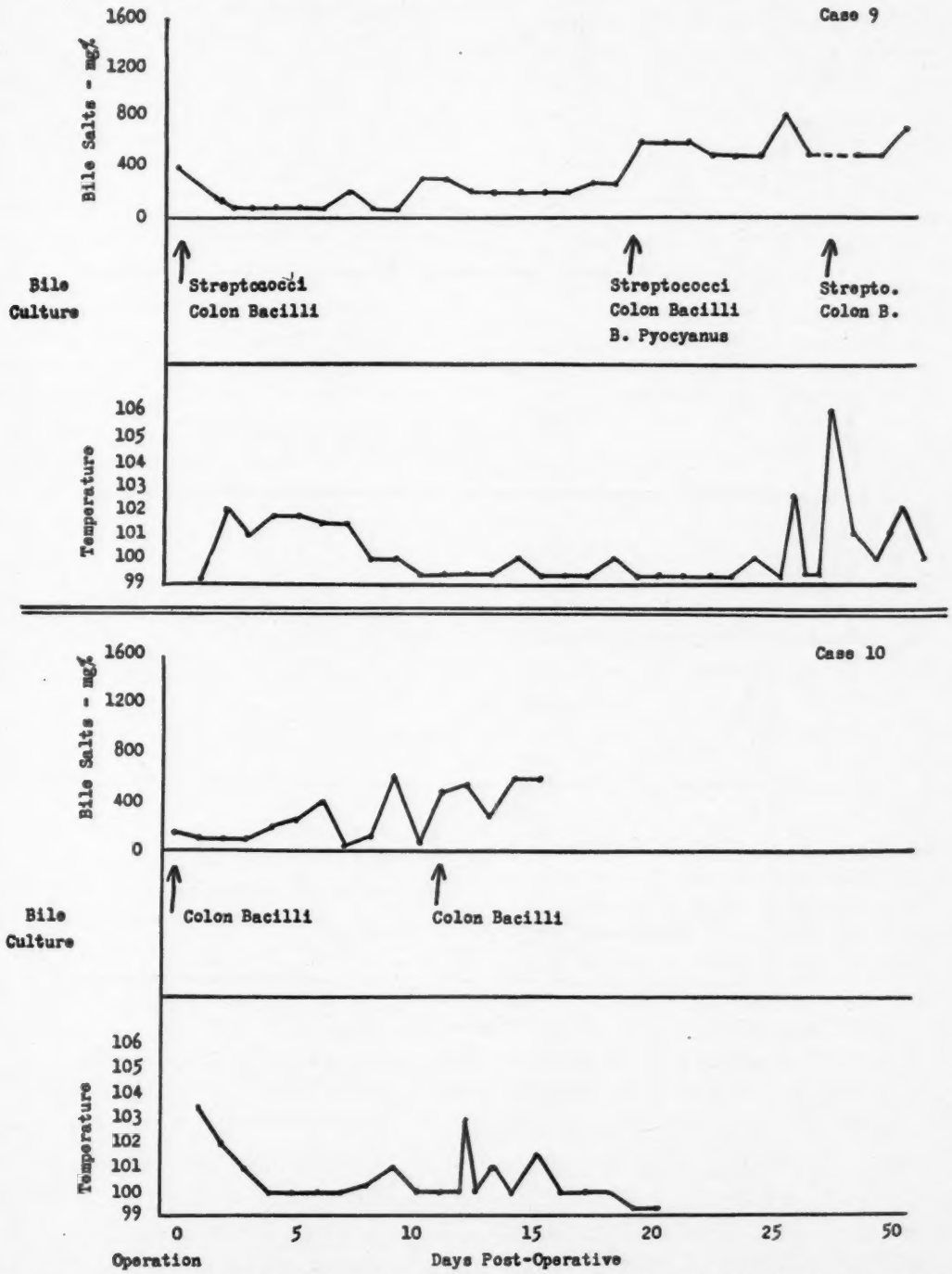

Fig. 5. The postoperative concentration of bile salts in two patients with an active purulent cholangeitis.

ANESTHESIA AND OPERATIVE TRAUMA

The initial postoperative depression in the concentration of bile salts noted in

anesthetic agents on the degree of the initial depression. Severe operative trauma and prolonged operating time, as well as

the anesthesia, cause a continuing postoperative depression in the concentration of bile salts from apparently normal livers.

Case XII (Fig. 6), however, had a more severe cholecystitis with pericholecystic adhesions and several stones in the common

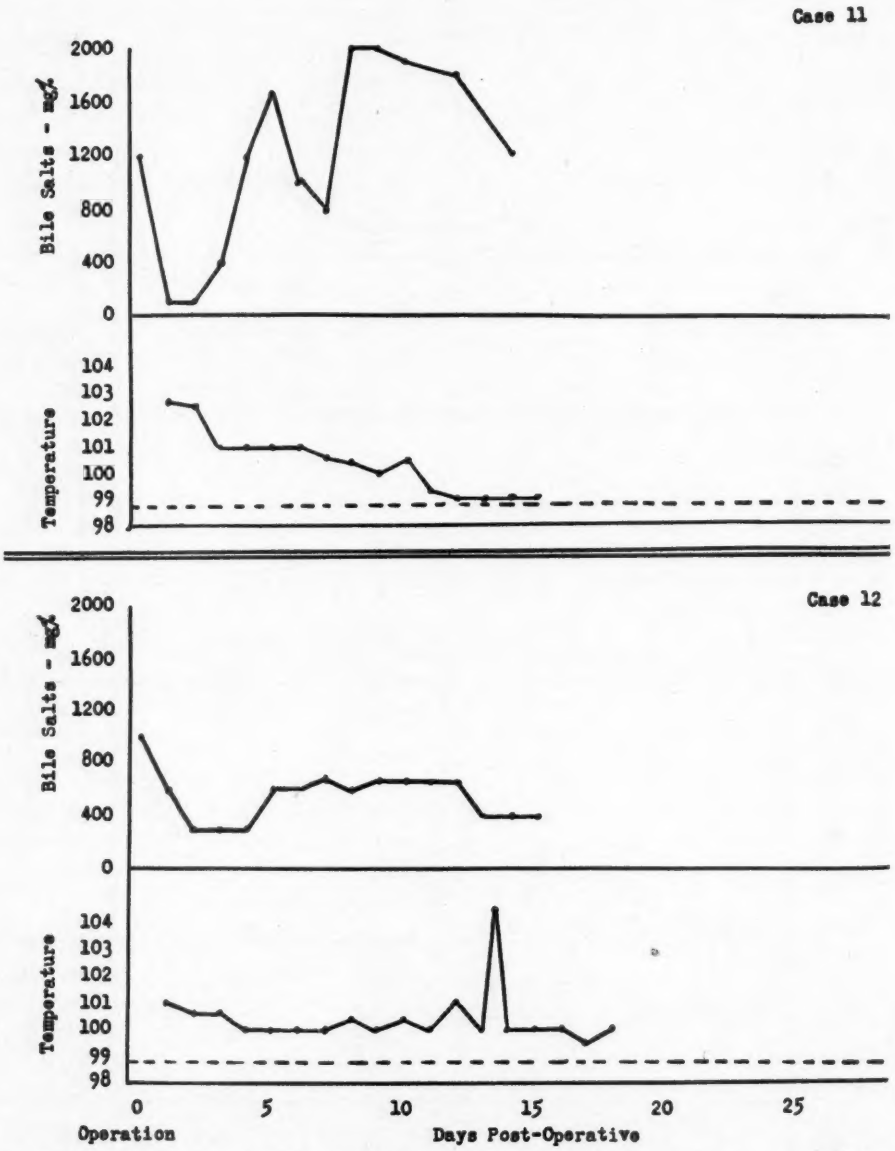

FIG. 6. The postoperative concentration of bile salts in two patients, one of whom (Case XI) had minimal operative trauma and the other (Case XII) severe operative trauma.

Case XI (Fig. 6) illustrates the immediate postoperative depression that is generally seen in fistula cases. The bile salt concentration had returned to normal by the fourth postoperative day. This patient had a chronic cholecystitis with a moderately dilated common duct so that cholecystectomy and choledochostomy were easily accomplished in forty-five minutes,

duct. In spite of this pathology, the liver appeared normal and was without evidence of obstruction. Cholecystectomy and choledochostomy required nearly two hours and were completed only after considerable trauma to the liver. It is probable that in the majority of cases the duration of the anesthesia and the operative trauma contribute as much to the postoperative

hepatic insufficiency and retard the rate of return to a normal hepatic function as does the choice of anesthetic.

GLUCOSE ADMINISTRATION

Many clinicians have noted the clinical improvement in jaundiced patients upon the parenteral administration of dextrose solution. A high carbohydrate diet, in patients with less severe jaundice, apparently improves the function of the liver. Eliason and Erb,[21] and Ravdin, Rhoades, Frazier and Ulin[22] have shown that such therapy has in recent years reduced the mortality of surgery on jaundiced patients. Gray and his co-workers observed no immediate effect of dextrose administration upon the concentration of bile salts in fistula cases. Our experience contradicts their findings, in that in eight of our cases in whom a secondary diminution of concentration was seen late in the drainage period, administration of dextrose in increased amounts has brought forth an increase in the concentration of bile within two hours. It does not necessarily follow that the dextrose was responsible for this response, but in Cases XIII and XIV such a relationship appears to have been established.

Case XIII. A 74 year old woman was admitted with a short history of right upper quadrant pain. A mild jaundice developed three weeks before admission and persisted. She had had a cholecystectomy thirty years before for calculous cholecystitis. The icteric index was 15 units, the Van den Bergh direct and the serum bilirubin below 2 mg. per cent.

Upon operation the common bile duct was markedly dilated and contained two calcium bilirubinate stones. The liver was granular in appearance but otherwise not remarkable. A choledochostomy was performed. The patient had a stormy postoperative convalescence.

She was given some parenteral dextrose for the first three days. This was discontinued and the patient placed on a soft diet. The bile salts diminished to only 50 mg. per cent by the sixth day, at which time intravenous dextrose was given with an immediate increase in concentration of bile salts. This treatment was continued up to the eleventh postoperative day. Upon discontinuing this therapy, the concentration of bile salts again diminished. It finally rose on the fourteenth day, probably the response to recovery of function by the liver. (Fig. 7.)

Case XIVA. A 60 year old woman had a history of sharp epigastric pain of six months' duration. She had no jaundice at any time. At operation an obliterated calculous cholecystitis was found. The liver appeared normal. There were two stones in the common duct, different from their fellows in the gall-bladder. Cholecystectomy and choledochostomy were performed. Convalescence was prolonged but uneventful. Figure 7 shows the concentration of bile salts in this patient during the immediate postoperative period. At no time was she given parenteral dextrose.

Case XIVB. Eighteen months later she returned with similar symptoms as well as jaundice of two weeks' duration. At this time her blood cholesterol was 205 mg. per cent, cholesterol esters 60 mg. per cent, icteric index 44 units, Van den Bergh direct and serum bilirubin 9.4 mg. per cent. Duodenal drainage showed a moderately concentrated bile containing calcium and calcium bilirubinate crystals and a pure culture of colon bacilli. She was prepared for operation by means of a high carbohydrate diet and daily infusions of dextrose solution. The concentration of bile salts over a period of three weeks is given in Figure 7. On the twenty-second postoperative day, after the severe secondary diminution in concentration we have previously described, a detailed hourly study was made of the concentration of bile salts following the use of intravenous dextrose. The result of this study is shown in Figure 8.

Discussion. In these two cases we have attempted to reduce the variables as much as possible in order to show the response of the liver in bile salt secretion to the administration of dextrose. In Case XIII, the patient, with moderate liver disease, had only a slight concentration of bile salts in the bile. On the administration of dextrose, this rose, only to decrease when this form of therapy was discontinued.

In Case XIV we have a better example of the response of a liver to the administration of dextrose. At her first operation this

patient received only routine care. The concentration of bile salts in her fistula bile was minimal throughout the twenty-four

the icterus was greater than on the first admission. She was prepared by the administration of adequate amounts of car-

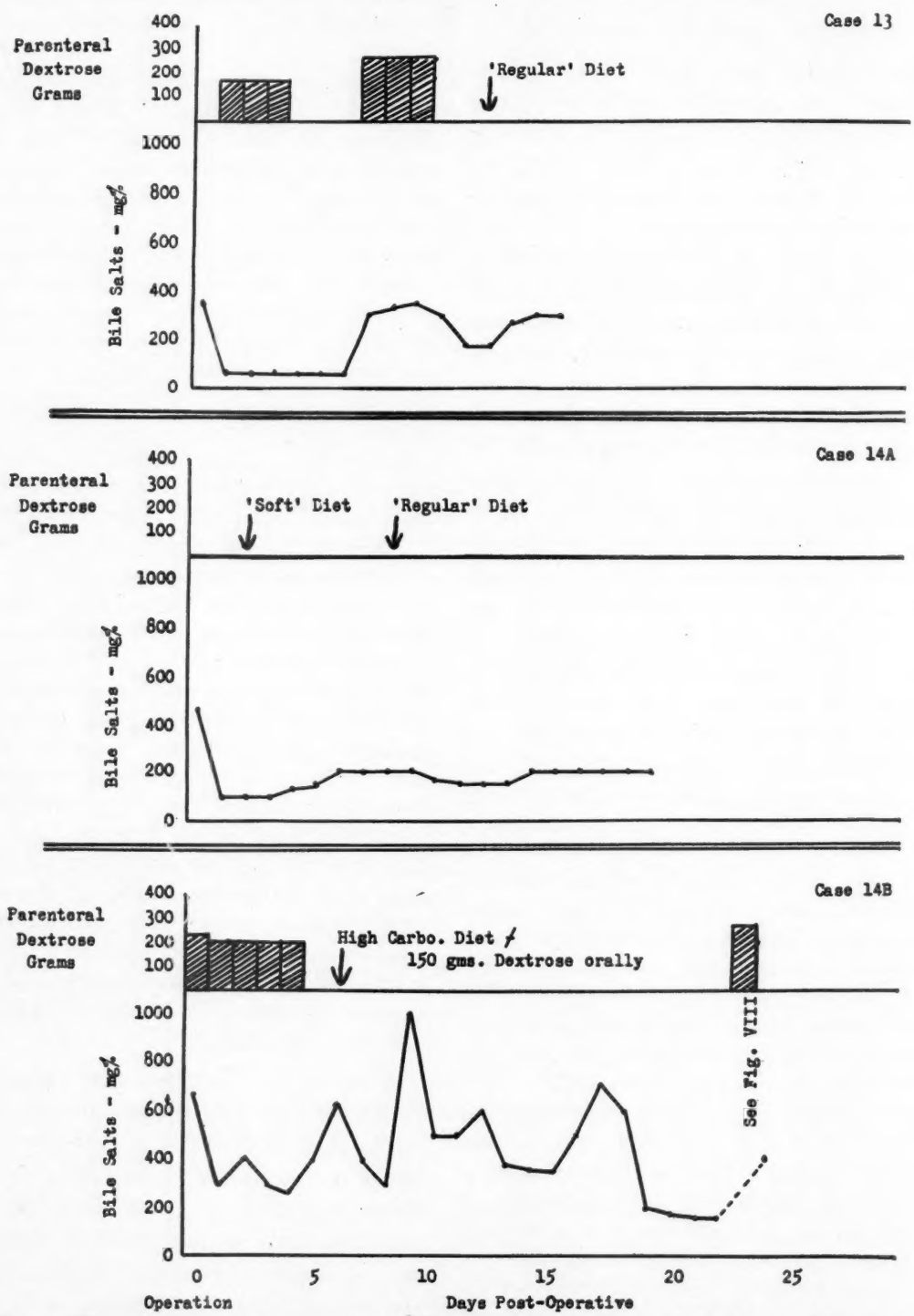

FIG. 7. The postoperative concentration of bile salts in cases showing the influence of dextrose administration on the degree of concentration.

days of study. When she returned for a second operation the degree of hepatic impairment measured by the intensity of

bohydrates. This preoperative preparation was reflected in the higher concentration of bile salts found in the bile at opera-

tion and secreted by the liver immediately after operation. When, on the nineteenth postoperative day the concentration of bile salts diminished, dextrose was given, with an immediate increase in the bile salt output. That this was no accidental finding is shown in Figure 9 on which the hourly determinations have been graphed. The experiment has eliminated to a large extent the many variables seen in these cases and

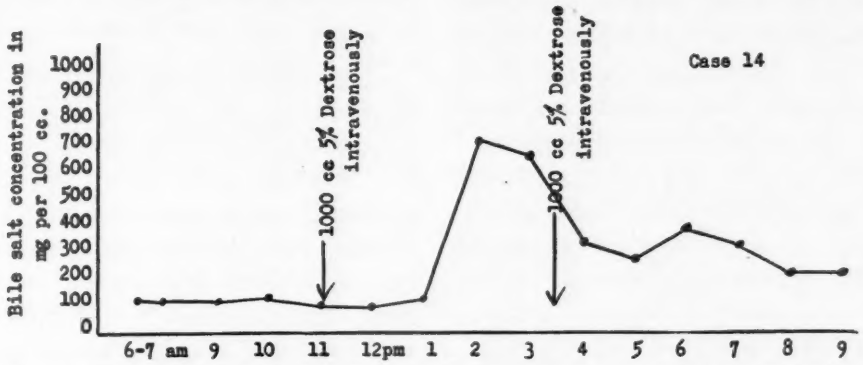

Fig. 8. The hourly determination of the concentration of bile salts following dextrose therapy in a patient 22 days postoperative.

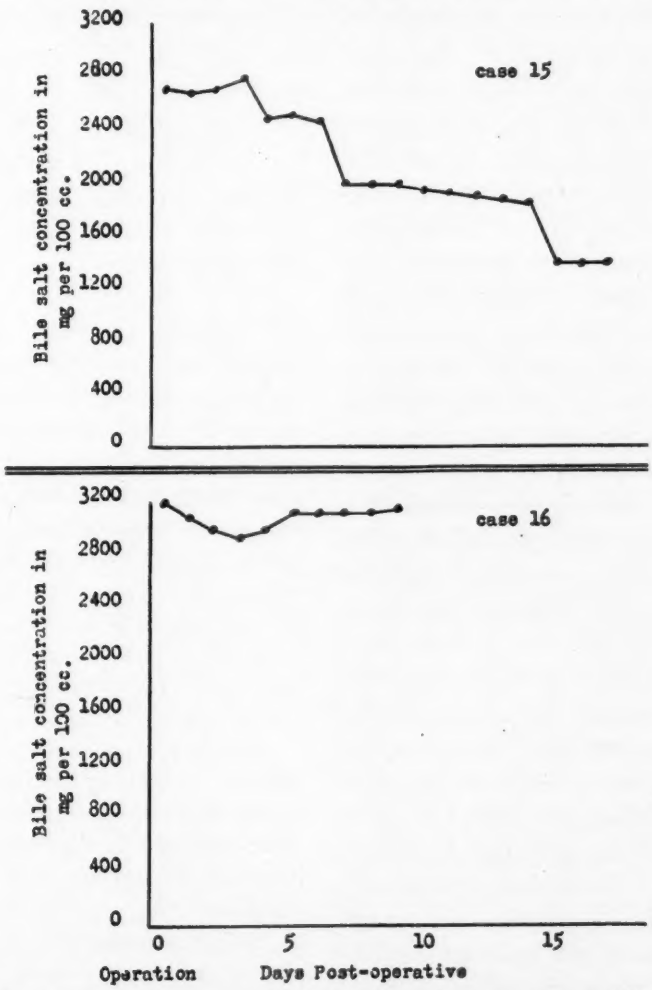

Fig. 9. The postoperative secretion of concentrated bile suggesting the concentrating action of the common bile duct in two patients with normal livers but with markedly dilated common bile ducts.

leaves little doubt that in this case the dextrose did improve the functional activity of the liver. Whipple reported that the bile salt content from fistula bile was reduced when dextrose was administered and thought this due to a conservation of the bile salts by the liver. The cases cited, as well as the results in fistula patients who were adequately prepared with dextrose preoperatively, indicate that the bile salt secreting function of the liver is improved by the administration of dextrose.

BILE AND BILE SALT ADMINISTRATION

Considerable study has been devoted to the value of the administration of bile salts to patients with biliary fistulae. Kohlstaedt and Helmer[11] are certain that the oral administration of bile salts increases the concentration of bile salts in fistula bile. They found that the ingestion of natural bile caused the greatest increase in concentration. Doubilet and Colp[23] in experiments on both humans and dogs, came to a similar conclusion. They believed that the salts of cholic acid were the most efficacious. The most satisfactory tests for bile salts in bile give only the cholic acid content and therefore the increase in total bile salt content cannot be accurately determined. Clara,[24] and Cantarow and Stewart,[25] found that dehydrocholic acid and its salts (decolin) act as a protective mechanism to the liver in obstructive disease of the extrahepatic biliary tract.

Mixed bile salts were administered to the cases in this series, usually beginning on the second or third postoperative day. In eight cases the fistula bile was re-administered to the patient by proctoclysis. No effect on the concentration of bile salts in the fistula bile could be noted in these patients though the general physical condition improved, the ability to take food increased, and flatulence and nausea decreased. Such improvement in the physical state of patients has also been reported by Ravdin, Riegel, Johnston and Morrison.[26] We could come to no conclusion concerning the efficacy of the administration of bile salts in increasing the concentration of bile salts in fistula bile.

CONCENTRATING ABILITY OF THE COMMON BILE DUCT

The ability of the common bile duct to concentrate the hepatic bile under certain abnormal conditions has been established by Schmidt and Ivy,[30] Sweet,[28] Boyd,[27] Sutton,[29] and others. The bile from a T-tube may be concentrated by the common bile duct and so not be true hepatic bile. This increase in the concentration of the bile will depend upon the length of time the bile is in the common duct before entering the T-tube and is directly dependent on the total volume of bile secreted and the size of the common duct. The concentrating ability of a dilated common duct must therefore be considered in interpreting the finding of concentrated bile from a T-tube fistula. Two case reports and chemical studies that illustrate this factor are presented.

Case xv. A male, age 50 years, was admitted with a short history of continuous epigastric distress for three months. One month after the onset, he noted jaundice which continued up to the time of operation. The blood cholesterol was 290 mg. per cent, the cholesterol esters 120 mg. per cent, the icteric index 70 units, the Van den Bergh direct, and the serum bilirubin 2.4 mg. per cent. Concentrated bile was secured on duodenal drainage. The duodenal bile contained calcium bilirubinate and cholesterol crystals. The gall-bladder could not be visualized roentgenologically.

At operation, the gall-bladder was found dilated, inflamed and filled with stones. The cystic duct was completely occluded. The liver was enlarged but of normal color and appearance. The common bile duct was dilated to a diameter of 3.5 cm. It contained no stones and a probe entered the duodenum with ease. A cholecystectomy and choledochostomy were done. The bile aspirated from the common duct at operation was markedly concentrated. Figure 9 shows the postoperative drainage findings in this patient.

Case xvi. A 67 year old male had had a cholecystectomy, ten years previous. He was

admitted with a history of epigastric pain of one year's duration. The pain radiated to both right and left upper quadrants. Jaundice appeared four days before admission. The blood cholesterol was 187 mg. per cent, the icteric index 100 units, the Van den Bergh direct and the serum bilirubin normal. Duodenal drainage showed a concentrated bile containing clumped calcium bilirubinate crystals and colon bacilli.

Upon operation, the common bile duct was dilated. It contained calcium bilirubinate stones. The bile was concentrated (3,070 mg. per cent bile salts) and cultures were positive for colon bacilli. The liver was enlarged, discolored and granular. After removal of the stones, the duct was found to be patent. Figure 9 gives the bile salt concentration of the fistula bile for the first eight days. The T-tube was inadvertently removed at this time, but in spite of this the biliary sinus healed in three days and the patient made an uneventful recovery.

Discussion. It is difficult to correlate the findings in these two patients with those seen in other patients with apparently normal livers. The high concentration of bile salts in the operative bile and the continuation of such high concentrations postoperatively is presumptive evidence that the common bile ducts of these patients had the ability to concentrate the bile. It may be argued that the secretion of the liver was so concentrated but analysis of the normal cases fails to reveal hepatic bile with a concentration over 1600 mg. per cent unless the common bile duct was markedly dilated. Further evidence of the ability of the common bile duct to concentrate bile salts is given in the fact that on clamping the T-tube the concentration increased. It is, therefore, probable that in the interpretation of any study of the concentration of bile salts from the liver, the ability of the common bile duct to concentrate the bile must be considered.

SUMMARY

The postoperative changes in the concentration of bile salts in the bile have been studied in a series of surgical patients following drainage of the common bile duct through a T-tube. When the patient has had no evidence of hepatic disease or the latter is minimal, there is a temporary reduction in the concentration of bile salts in the bile, followed after two to three days by a progressive return to normal levels. This drop is interpreted as due to such factors as the type and duration of anesthesia, the local and constitutional effects of operative trauma, the degree of preoperative biliary obstruction with hydrohepatosis, and the like.

The rapidity of the postoperative return toward a normal concentration of the bile salts in the bile and the maximal concentration attained during the period of observation in general are inversely proportional to the degree of hepatic damage. Evidence is presented that such factors as systemic infection, cholangitis, depletion of bile salts from prolonged drainage, and an inadequate supply of carbohydrate will reduce the concentration of bile salts in the bile, presumably as a result of functional as contrasted to structural changes.

The multiplicity of factors which apparently affect the functional ability of the liver and so the concentration of bile salts in the bile correspondingly increase the difficulty in determining the factors responsible for the changes in any individual case.

Evidence was obtained suggesting that in some instances the common bile duct may concentrate the bile passing through it in the same manner as is done in a normal gall-bladder.

The continued failure of the liver to secrete bile salts in the bile is evidence of severe functional disturbance and so of serious prognostic import.

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WHEN TO OPERATE AND WHY, AND WHAT OPERATION TO DO IN ACUTE CHOLECYSTITIS*

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THIS presentation is founded on records of over 1500 personally operated cases covering many years and on many more cases in which operation was not advised. Conclusions have been arrived at from a study of not only the immediate mortality but also the morbidity results.

Acute cholecystitis is but one type of gall-bladder disease and forms a very small proportion of all gall-bladder cases. Gall-bladder disease itself is a chronic, slowly progressive inflammation with a large incidence in relation to population, beginning in young adult life and gradually increasing in severity with markedly variable clinical manifestations, the latter so variable as many times to pass as negligible factors. The disease is subject in a large proportion of cases to long periods of latency of clinical manifestations and in a small proportion of cases to sudden exacerbations of acute inflammation. In the course of the disease the function of the liver is affected to a variable degree. Nearby organs are affected through lymphatic supply (pancreatitis, localized hepatitis); through contiguity (adhesions, perforation); and through mechanical affects (pancreatic duct). Distant organs and mentality are affected through liver disturbance (jaundice, impaired liver function) and through the gall-bladder, acting as a focal infecting agent (kidney lesions, heart lesions, joint lesions). The disease is not only widespread, but has limitless potentialities.

Incidence. Our experience leads us to believe that gall-bladder disease is even more wide spread than was formerly supposed, for the following reasons: It is only in the last two decades that the knowledge of gall-bladder pathology has been sufficiently well known to permit us to consider autopsy findings as minimum rather than maximum figures. Moreover, even now the gall-bladder which I described in 1916 as cholecystitis catarrhalis subacuta, the first stage in gall-bladder disease, is perhaps not recognized through its gross pathologic appearance by any except the expert.

The Proportion of the Disease Which Is Surgical. Considering gall-bladder disease as defined, only a very small percentage of cases require surgical intervention. It is estimated that of every 100 cases eighty either become quiescent or present such minor disturbances as not to interfere very much with the patient's well-being and therefore do not require surgical intervention. Of the remaining twenty there are perhaps fifteen in which operation is distinctly indicated and five in which it is a question of judgment as to whether or not operation should be performed. Of the cases which in all probability will require surgery at some time or other, the proportion in which immediate surgery is necessary is quite small.

Medical treatment is indicated in all acute inflammations of the gall-bladder showing a tendency to subside, with the exception of acute cholecystitis with gangrene, perforation, empyema and cholangitis. Medical treatment is also indicated in chronic cholecystitis tending toward latency and in hydrops of the gall-bladder (unless the tumor produces pressure symptoms or worries the patient) and in cholecystitis cicatrans not giving pain. Medical treatment is indicated in the acute cases

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because they are usually temporary, and in both acute and chronic because they are not disabling, do not give the patient much discomfort and do not interfere to any great extent with his enjoyment of life and his ability to earn his living.

Per contra, surgical treatment is indicated in acute cholecystitis which does not subside quickly, cholangitis both acute and chronic, gangrene of the gall-bladder, perforation of the gall bladder, beginning carcinoma of the gall-bladder, in all cases of chronic cholecystitis (of which there are many forms) in which health is affected over a period of time through recurring attacks of pain and where comfortable digestion and the pursuit of a livelihood are definitely impaired.

We differentiate between two classes of surgical cases: those in which operation must be done at once in order to save life; and those in which we have time to consider whether operation is advisable. In the first group the physician and surgeon must throw the weight of their experience in the balance to influence the patient's decision; they must insist upon immediate operation. In the second class there is time—the case may be studied and discussed with the patient and a mutual decision is arrived at in accordance with the degree of interference with the patient's comfort and earning power.

In the first class are the cases of acute cholecystitis not rapidly subsiding, the cases of seropurolent chronic cholangitis, acute cholangitis, gangrene of the gall-bladder. In the second class there are cases of chronic cholecystitis in which the patient's comfort and occupation are being materially affected. In the second group it is really up to the patient to give the final decision. We can, however, lay before such a patient his probable chances for cure or at least for the decided amelioration of his symptoms by operation and also the mortality of such an operation in skilled hands.

The desirability of tiding over the acute cases is indicated by 545 operations for acute conditions with a resultant mortality of 10.3 per cent compared with 661 operations for chronic conditions with a mortality of 2.3 per cent. Both series include common duct involvement.

Very few cases of acute cholecystitis are *immediately* brought to the surgeon, so to speak of operation as *immediate* is not quite correct. These patients are seen two or three days, sometimes a week, after the initial symptoms. Patients seen very early, i.e., within twelve hours of the beginning of an acute attack, are generally treated like cases of acute appendicitis; immediate operation is done, sometimes because the case is improperly diagnosed as acute appendicitis and the real lesion is discovered only at operation. Such patients usually do very well both as to mortality and morbidity. The cases seen at a somewhat later date and in which the diagnosis is properly made, which may be called *delayed operated cases* (necessarily delayed because they were not brought to the surgeon in the very inception of the attack), may or may not be better operated upon immediately. It is my custom to delay operation for a few hours until it can be determined whether the disease is progressive. If so, so-called immediate operation is done; if not, the patients are carried over to a period of quiescence. Otherwise, we might find the pathology so acute and extensive as to cause undue mortality in what should be the operation of election, namely cholecystectomy (278 acute cases with a mortality of 6.5 per cent). If the case can be tided over to a period of quiescence, then this procedure may be done much more safely (589 operations in chronic cases with a mortality of 1.9 per cent).

If immediate operation is necessary, the type of operation will depend upon the pathology present and the acuteness of the inflammation. Cholecystectomy should be done if this is possible without undue risk (6.5 per cent mortality as compared in 278 cholecystectomies with 235 cholecystostomies in acute cases with a mortality

of 12.8 per cent; the cholecystostomies presented very acute and extensive pathology). In deciding between these operations, I am guided by the amount of inflammation at the base of the liver as shown by inflammatory thickening and adenitis surrounding the cystic duct and the site of emergence of the hepatic ducts from the liver. When this inflammation is intense and massive I prefer cholecystostomy as being the safer. One must also consider the presence or absence of jaundice. If it is absent, either method may be indicated; if jaundice is present, an exploration of the common duct is in order, either by touch or by incision and exploration. It is to be recalled that very frequently the jaundice which we find at times associated with acute cholecystitis is due to pressure on the common duct or hepatic ducts by the adenitis at the base of the liver and not to intraduct obstruction. I rarely use drainage in cholecystostomies in acute cases, aside from tube drainage of the gall-bladder, unless common duct drainage is also done. In acute cholecystitis where the gall-bladder has been removed, I usually use a cigarette drain to the foramen of Winslow. A comparison of 543 cholecystectomies with drainage and 240 without drainage showed that both mortality and morbidity were better in drained cases.

Morbidity in these procedures, excluding choledochostomies, cholecystenterostomies, cholecystgastrostomies and choledochoplasties, has been studied in the postoperative records of 979 cases for three to twenty-five years. Those patients relieved of all symptoms are called well, those who have an occasional slight indigestion or suffer at times from over-eating are classed as having mild symptoms. Of 165 cholecystostomies, 58.1 per cent were well and 14.5 had mild occasional disturbances of digestion; that is, 72.7 per cent were practically well. Of 814 cholecystectomies, 86.6 per cent were well and 9.7 per cent showed mild symptoms; that is, 96.3 per cent were practically well. These good results would have been much better had the patients been operated on before the liver was damaged so extensively as to require treatment even after the infecting focus was removed, and before the inflammation had resulted in adhesions, stricture of ducts, perforation, gangrene, peritonitis, cholangitis, liver insufficiency, hepatitis, liver abscess; distant infections from the local focus such as myocarditis, renal insufficiency, pulmonary infections, joint complications; involvement of neighboring organs through extension of the inflammation such as pancreatitis, perforation into the stomach, duodenum or colon, adenitis causing pressure on the common duct either beneath the liver or at or near the ampulla of Vater; carcinoma of the gall-bladder following long-continued chronic irritation, to say nothing of the discomfort and disability from the at first localized disease.

SUMMARY

It has been the purpose of this paper to show what acute cases should be operated on, what is the best time for operation, what type of operation is safest, and what operation gives the best final result.

OVARIAN NEOPLASMS: THEIR PATHOLOGIC AND SURGICAL SIGNIFICANCE*

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OVARIAN neoplasms belong to the group of most common lesions encountered in the practice of medicine and surgery. Some are more important than others, yet not one, even if it is benign, can exist for very long without the loss of ovarian function and finally complete destruction of the organ. The results of treatment of both the benign and the malignant types are often disappointing. In addition, the pathologic characteristics of these neoplasms, it seems to me, have not been sufficiently stressed. A clear conception of the type of tissue being dealt with is the fundamental basis for the application of correct surgical principles in treatment.

The ovary is one of the most complex organs in the human body in that it not only is an organ of reproduction but also produces hormones of internal secretion which profoundly regulate many physiologic processes of the female. Many of the functions of the ovary are known but I am certain that others are not known and are not even suspected. These probably will not be discovered until more intense study is made in the physiologic chemistry of the various cells in the ovary. In this connection it is of interest that only recently a close relation between calcium metabolism in muscle and ovarian function was reported. That factors other than reproduction are related to the ovary is shown by the fact that cancer of the breast is rare among castrated women. Cancer of the body of the uterus has likewise not been encountered among patients whose ovaries have been removed before the onset of the natural menopause. This does not hold true for patients who have been given radium or Roentgen therapy sufficient to produce a menopause, but in whom the ovary has not been removed or completely destroyed. At present, no one knows how much irradiation is required to produce the same effect as bilateral oöphorectomy. These observations are cited merely to stimulate interest in the complexity of the functions of the ovary and to emphasize that lesions of the ovary, whether benign or malignant, should receive more careful consideration.

Surgical treatment of ovarian lesions should be undertaken with meticulous care and only after due consideration. The indiscriminate removal of the ovary for simple cystic conditions or for so-called ovarian pain whether proved or not, should be condemned.

The consistency of ovarian tissue in different individuals has a great range of normal variation as to size, irregularities of the surface, thickness of the cortex and the number of retained follicular cysts. Also, the appearance of the organ varies tremendously according to the phase of the menstrual cycle. For example, there may be a blood clot of moderate size adjacent or adherent to a corpus luteum; yet this may be entirely normal. Occasionally both ovaries may be slightly elongated and irregular as a result of retained follicles, and yet be normal for the particular patient, who has, very likely, sought treatment for periodic attacks of pain in the right and left lower abdominal quadrants. In most instances of this type attacks are due to rupture of a retention cyst or occasionally to slight oozing of blood from the wall of the cyst. Not infrequently such patients are submitted to an appendectomy and if

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this does not give evidence of acute disease, the ovary often is removed as the cause of the pain.

For some uncertain reason the ovary may never function properly, or if once it has functioned normally, its function may become arrested. Yet to all gross appearances it is normal. Occasionally this derangement is secondary to faulty function of some other organ closely related to the generative organs; for example, the thyroid gland. This condition during the third decade of life is a very real problem to the patient and to the physician. Young women in this group are subjected to many varied types of therapy; some respond well to proper management whereas some do not.

Radium therapy has been prescribed with the idea of producing temporary abeyance of ovarian function, thereby controlling uterine hemorrhage. This procedure brings about in a young woman a temporary artificial menopause which seems to me absolutely contraindicated. If the bleeding cannot be controlled by any type of substitution therapy in an otherwise normal individual, I am of the opinion that abdominal hysterectomy with preservation of the ovaries is justified and is the logical surgical treatment. It is true that the ovaries sometimes become cystic and require surgical removal, but not all of them will require it by any means. The percentage of survival of the ovaries after hysterectomy is determined for the most part by the extent of the interference with their blood supply. The ovarian branches of the uterine arteries usually are destroyed, but the main ovarian artery and vein are not disturbed. However, if these vessels in the ovarian pedicle are left under considerable tension the vessels will gradually become thrombosed and the ovary as a result will become cystic or atrophic.

It is important to distinguish between menstrual disturbances that arise from abnormal function of the ovary and those attending ovarian neoplasms. The histologic picture of the endometrium in both instances may be identical and in cases of functional bleeding alone there is no objective evidence of lesions of the ovary. Neoplasms usually disturb normal ovarian function but not in every instance and not always to the same extent. To understand the ovarian disturbance in the endometrium it is necessary to be familiar with the physiologic and pathologic variations in the endometrium.[2]

Briefly it is sufficient to say that the endometrium is the most active tissue in the human body and that there is no so-called resting stage. In normal conditions the endometrium completely changes itself every twenty-eight days. The first fourteen days are designated as the proliferative phase, changes being brought about through the action of the follicle. The last fourteen days are designated the differentiative stage, controlled by the corpus luteum. Whenever failure of formation of the corpus luteum supervenes or if there is an excessive follicular effect, the endometrium becomes cystic. This has not been sufficiently stressed; it deserves attention since it denotes ovarian failure from some cause. A cystic endometrium in the past has been alluded to as hyperplastic, although it is not hyperplastic at all, but on the contrary, is evidence of degeneration.

One other point of extreme importance in this connection is that activity of the endometrium may be arrested at any point in the menstrual cycle; therefore, it may be designated pathologically as the persistent proliferative or persistent differentiative phase of the menstrual cycle, resulting in periodic menstrual bleeding or periodic amenorrhea.

A correlation of these findings in the endometrium with those of benign ovarian neoplasms, such as cystadenoma and adenomyoma, is often advantageous in planning surgical treatment. The cystadenoma is an outstanding example of a benign ovarian neoplasm. It arises in the substance of the ovary in contradistinction to the simple follicular cyst, and so when, as frequently happens, it reaches considerable size, the ovary usually is destroyed. Second, these neoplasms may be unilateral but unfortu-

nately a high percentage are bilateral, if not at the time of operation then later in life. If the endometrium reveals beginning ovarian failure with menstrual irregularities associated with a benign cystadenoma of one ovary, there are most likely small multiloculated cysts within the substance of the remaining ovary and the prognosis for correction of the disturbances of menstruation by removal of the one cystadenoma is not good. On the other hand, if the endometrium does not show degenerative changes the prognosis should be excellent. A large ovarian tumor that completely fills the abdomen of a young woman is usually of this type. It is not malignant, in contradistinction to the papillary cystadenoma which is malignant.

I wish to place considerable emphasis on the word "papillary" in this connection. I feel that it is pathologically sound to regard and to treat as malignant any tumor which results in the formation of papillary epithelium. To do otherwise is to court disaster. Papillary epithelium behaves in characteristic fashion regardless of where it is situated. For example, if it appears in the urinary bladder it may or may not be quiescent but if it is disregarded, eventually it will kill the patient.

Endometriosis (adenomyoma) is another benign lesion of the ovary important because of its frequency, its appearance in early adult life, its effect on sterility, the physical disability and the disturbance of ovarian function attending it. This lesion is not confined to the ovary, but the activity of any associated adenomyomas is entirely dependent on the ovary. Roentgen rays or radium therapy sufficient to bring about arrest of ovarian function will render all lesions inactive unless the ovary partially regains its function after such treatment.

The disturbance of ovarian function in this type of disease is a factor which is in need of very careful study. It has been my belief for considerable time that endometriosis of itself does not affect, to any appreciable degree at least, the function of the ovary, since most patients who have this condition have associated lesions of the uterus or ovary which could sufficiently explain the menstrual irregularities. In a recent series of 308 cases I discovered that in 65.6 per cent menorrhagia, metrorrhagia or both occurred; this was approximately the same as the number of associated pathologic lesions of the uterus and ovary. However, the endometrial lesion may be the chief factor of the menstrual disturbance or an incident in a sequence of events resulting from ovarian failure. Some evidence which would tend to clarify this point might be found in a study of the incidence and distribution of endometrial lesions in cases of so-called functional uterine bleeding.

With regard to the effect on ovarian function, I have recently been impressed with the high incidence of cystic changes in the endometrium, ranging from a mild to a severe degree, among patients who have been subjected to operation primarily for endometriosis. Furthermore, the extent of cystic change seems to bear a definite relation to the amount of endometrial disease and hemorrhagic cysts of the ovary.

There is considerable disagreement among students of this subject in regard to whether hemorrhagic cysts (chocolate cysts) of the ovary are the result of endometrial ovarian lesions. Certainly in many cases of large hemorrhagic cysts there is no pathologic evidence of endometriosis in the ovary or wall of the cyst but endometrial lesions usually are identifiable on the posterior surface of the broad ligament adjacent to the ovary, although certainly not in great amount. When endometriosis is the outstanding abnormal condition present, the hemorrhagic cysts are usually very small but often multiple. Some of the cysts contain blood of recent origin and in some it is tarry as a result of decomposition of long standing.

We have observed also that when endometriosis is the predominant abnormal condition present, dysmenorrhea is the prnicipal symptom, but when chocolate cysts are predominant, menstrual disturbance is the chief symptom. Pain and menstrual disturbance together usually denote

rather extensive involvement by both types of lesions.

The treatment of these lesions seems to me to be primarily surgical unless the rectovaginal septum is involved extensively by endometrioma, in which case radium therapy sufficient to arrest ovarian function would involve considerably less risk. However, if the pelvis is filled with large chocolate cysts it is wiser to perform bilateral oöphorectomy and leave the lesion in the rectovaginal septum undisturbed. In the case of a young woman who suffers predominantly from these lesions, the best treatment is to excise the involved structures and preserve the menstrual or the reproductive function or both. Such procedures are indicated only for patients whose lesions are not extensive or are confined only to one adnexal region because the incidence of recurring symptoms in cases of more extensive involvement is quite high. Sterility, of course, is a prominent complaint and is often the symptom which induces the patient to seek medical advice. There is associated with this disease an absolute sterility in about 32 per cent of cases, but to correct it surgically is not always possible. In 56.2 per cent of a group of patients who had been treated by conservative surgical measures pregnancy occurred, but 16.8 per cent of these pregnancies resulted in miscarriages. Hope is eternal in some women with regard to reproduction and if they can live under the impression that they can reproduce they have much more peace of mind.

If the evidence of ovarian failure is strong, as exemplified by menstrual irregularities and cystic endometrium, I feel that the patient will be better cared for by a subtotal hysterectomy and by excision of the ovarian lesions, without complete ablation of ovarian function. It has been my observation that the activity of small lesions which may remain in the pelvis is considerably reduced if the patient does not menstruate, although the ovarian tissue remains.

Radical treatment is required for extensive lesions of this type which excessively involve the ovaries of patients who are in the latter part of the fourth decade of life. Also, considerable subacute pelvic distress due either to dacterial invasion or to the chemical action of blood pigment is more safely eliminated in this manner.

The significance of malignant lesions of the ovary is such that the question of preservation of ovarian function must be disregarded except in some unilateral carcinomatous cysts of a low grade of malignancy. It is unusual to find a malignant cyst in a woman under 35, but it does occur sufficiently often to keep one constantly looking for it. The malignant cysts which occur in the ovary are carcinomatous cystadenomas, papillary, nonpapillary or mixed. From the standpoint of pathology these cysts should not be such a serious matter, because the majority grow very slowly and often remain confined to the ovary for months. Unfortunately a great number develop after the menopause and the only associated symptom is enlargement of the abdomen. If they occur during the menopausal period, the menstrual irregularity is regarded too frequently as a normal process. Later when the cysts reach huge proportions the cyst wall becomes thinned, especially at the sites of greatest pressure, and finally becomes perforated. The malignant process then rapidly spreads and the condition approaches the inoperable stage.

Complete surgical removal of all pelvic organs is the procedure of choice for patients 40 years of age or older, even if the cyst is unilateral. However, 50 per cent are bilateral at the time of operation. If the cyst has become perforated and there are secondary implants these may be held in check by Roentgen therapy.

True carcinomas of the ovary are of two types, the papillary adenocarcinoma and the solid adenocarcinoma. In this group of cases symptoms frequently are absent during the early or favorable operative period. Some of these growths can be discovered, however, by routine pelvic examinations, and often the patient is amazed to learn that there is something wrong with

her pelvic organs. Papillary adenocarcinomas originate as a rule from the surface of the ovary and, unfortunately, are of a high grade of malignancy. Since they extend from the surface of the ovary, they become attached early to adjacent structures and shortly assume such proportions that they become inoperable. Fluid in the abdomen is the rule and extension to other structures occurs in a few months. It is known that the prognosis is generally poor for cancers of this type, although many of the secondary growths become quiescent, as is the case in malignant cysts, if the parent growth is removed and the procedure is followed by the use of radium and Roentgen therapy. In my experience, the longest duration of life in such a case after this method of treatment was twenty years. It is important to note also that there is a high incidence of bilateral involvement, so that it is advisable to remove all the pelvic generative organs even if the growth is confined to only one ovary.

Solid carcinomas of the ovary represent a much smaller group than papillary adenocarcinomas, and they also vary in their rate of growth. Their presence is usually not suspected, and consequently there is a high incidence of pelvic attachment and extension to distant lymph nodes. Fluid in the abdomen is a frequent finding, and an undesirable one in relation to prognosis. The seriousness of this group of cancers was revealed in a recent study which showed that of the patients who had solid carcinoma, grade 3 or 4, more than 75 per cent succumbed within a few months due to recurrences.

The method of treating ovarian cancer is early surgical removal followed by radium and Roentgen therapy. In some cases of carcinoma, grade 1, confined to the ovary, irradiation is omitted. In many cases the lesions are definitely not amenable to surgical treatment but are treated, if the patient's condition will permit, by intensive radium and Roentgen therapy. Some time ago, I[2] investigated a recent series of 143 patients at the clinic. These patients had cancer of the ovary and were treated with surgical procedures alone; 65.4 per cent of them were living five or more years after treatment. One hundred and eighteen patients received intensive irradiation in addition to surgical treatment and of this number 50.5 per cent were living five or more years later. Thirty-six patients had extensive lesions and were suitable for irradiation only; 16.7 per cent of them lived five years or more after such treatment.

UNUSUAL NEOPLASMS

Another small group of infrequent ovarian neoplasms must be considered in this connection. The first of these is the arrhenoblastoma, or masculinizing tumor of the ovary. The clinical syndrome which follows the onset of this tumor is a change toward a male type of habitus and a development of the secondary sexual characteristics of the male. The main physical features of the Cushing syndrome are absent. These growths are recognized easily both from the surgical and pathologic standpoints. If a patient who has previously had normal menstruation, experiences the slow development of amenorrhea plus the distribution of hair similar to that of males, enlargement of the clitoris and changes of the voice to the masculine type, the possibility that an arrhenoblastoma is present is indicated. Pathologically the tumor closely resembles the seminiferous tubules of the testis. For this reason it was first called "adenoma tubulare testiculare." It is of interest that this tumor varies in its differentiation from normal testicular tissue to a type that is hardly recognized as such. These cells secrete a hormone which influences secondary sexual characteristics in a masculine direction. Furthermore, the tumors that have the least resemblance pathologically to testis show the greatest masculinizing effect. It follows, therefore, that when these tumors are highly differentiated the masculine effect is greatly reduced and often unnoticed clinically.

This neoplasm frequently is classed as malignant but very rarely has metastasis been noted. If present, metastatic lesions

can be destroyed or rendered inactive by Roentgen therapy as the cells are very sensitive to Roentgen rays. The lesion is always unilateral and, since it extends but rarely beyond its initial site of origin, cure can be obtained by local excision. This is of especial importance because these tumors are seen during the menstrual life of the patient.

Another very interesting tumor is the granulosa cell tumor or feminizing tumor. This hardly can be said to be a rare tumor as about 400 have been reported. Sixty per cent of these tumors occur after the menopause and 30 per cent between puberty and the menopause; 5 to 10 per cent occur before the age of adolescence. The influence of these tumors differs for the three age groups just mentioned. For example, among young individuals the tumor produces precocious menstruation with early sexual and somatic development. In middle life the tumor produces either amenorrhea of itself or amenorrhea followed by profuse and continuous menses. However, after the menopause, this neoplasm gives rise to a periodic pseudomenstrual type of bleeding in 90 per cent of cases.

The syndromes produced at the various ages are explained by the effects of the large quantity of estrin elaborated by these tumors. This hormone can be identified in both the blood and urine of patients who have granulosa-cell neoplasm and in almost unbelievable amounts in extracts of the tumor tissue. Its effect on the uterus is exactly what one would expect from excessive activity of estrin—hyperplasia of the myometrium and a thick, boggy, proliferative type of endometrium, usually containing cysts. Why the effect of estrin among young people is different from its effect on patients of more advanced ages is not clearly understood.

Pathologically, 90 per cent of these tumors are said to be malignant but of a very low grade; therefore they rarely extend beyond the local initial site of origin. About 90 per cent of the lesions are unilateral and they are usually classified as solid tumors. Different cell types are recognized but are not of particular interest.

Since these tumors are usually unilateral and are of a low grade of malignancy, conservative surgical measures can be safely and logically followed. After the menopause, however, panhysterectomy is usually in order.

The next two tumors of special interest are the dysgerminoma and Brenner's tumor, neither of which produces a hormone. The dysgerminoma is a very interesting lesion because, although pathologically malignant, clinically it runs a very benign course. This tumor is said to correspond to the seminoma of the testis with the exception that the latter behaves as its degree of malignancy would indicate. The tumor usually occurs in girls and young women who are less than 20 years of age. It is frequently bilateral and is associated with hypoplasia of the genital organs.

It is said that this is the most common lesion observed in pseudohermaphrodites and in individuals who have poorly developed gonads. It may be best classified as occupying a "neutral" position between the masculinizing arrhenoblastoma and the granulosa cell feminizing tumor. Although this lesion appears to be of a high grade of malignancy, cure is obtained by removal of the affected adnexa. When both ovaries are involved (35 per cent) complete hysterectomy with removal of both adnexa is indicated.

Brenner's tumor is a solid, non-hormone-producing tumor of the ovary although it may be found in association with ovarian cysts. It is characterized by slow growth and is similar to ovarian fibromas in lacking specific symptomatology. It resembles somewhat a granulosa cell tumor and microscopically it resembles a metastatic epithelioma. Surgical removal effects complete cure.

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HYSTEROSTOMATOMY: DUHRSSEN'S INCISION*

A PRELIMINARY STUDY

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HYSTEROSTOMATOMY or Dührssen's incision of the cervix has been employed to a greater extent in recent years in a growing group of obstetrical cases which have tried the patience and judgment of many obstetricians. These cases present urgent indications for immediate delivery; yet the cervix is incompletely dilated and offers the principal obstruction to delivery. In some of these cases cesarean section has become contraindicated because of the risk to the mother due to prolonged labor, ruptured membranes and potential infection.

In former years the indications for hysterostomatomy were limited usually to two: (1) in the elderly primipara, and (2) in eclampsia. Forceful manual dilatation of the cervix is no longer considered a good procedure since it results in the tearing of the cervix, hemorrhage, shock and high morbidity. Cutting of the cervix, on the other hand, produces a clean surgical incision and eliminates the danger of shock.

Indications. In general the indications for hysterostomatomy are well expressed by Dr. F. L. Adair, as quoted by Hunt and McGee:[1] "Dührssen's incisions are permissable only in those cases where prompt termination of labor is in the interest of either the fetus, the mother, or both. The cervix must be adequately effaced though it is improperly dilated, and conditions must be such that the subsequent operation for delivery would be properly performed if the cervix were already fully effaced and dilated. It is an operation for the specially trained practitioners, to be carried out only in a suitable environment such as a well-equipped maternity hospital."

As a rule the indications for this operation are multiple in many individual cases. The labors are generally unsatisfactory, with poor or ineffectual pains; the patient is in labor for many hours; the presentations are often other than the anterior occiput; the membranes have ruptured prematurely or have been ruptured for many hours; and maternal exhaustion or fetal distress is present although the cervix is incompletely dilated and immediate termination of labor is necessary. In many of the cases, despite frequent rest periods induced by sedation and the maintaining the mother's strength by intravenous fluids, labor progresses far too slowly and even comes to a standstill.

In our series of Dührssen's incisions performed at the University Hospital from 1935 to 1938, inclusive, we find that the indications may be classified as follows:

Maternal Indications: 1. Maternal exhaustion as a result of prolonged labor associated with primary or secondary uterine enertia and malpresentation of the head.

2. A true Bandl's contraction ring.

3. Pre-eclampsia with slow labor.

4. Labor coming to a standstill in an elderly primipara.

5. Previous cesarean section with thin uterine scar.

Fetal Indications: In any case where evidence of fetal distress is present, and where prolapsed cord is found.

Contraindications: It is generally agreed that placenta previa is an absolute contraindication for the operation since fatal uncontrollable hemorrhage may result

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from extensions into the enormous uterine sinuses.

SUMMARY OF FINDINGS

Age and Parity. Seventy-eight cases of hysterostomatomy were performed at the University Hospital from 1935 to 1938, inclusive. In this series, sixty-nine were essentially primigravidae; eight of this number gave a history of one previous abortion. The remaining nine patients were multigravidae. The ages of the primigravidae varied from 15 to 41 years with an average of 21.6 years; the ages of the multigravidae ranged from 16 to 38 years with an average age of 30.7 years.

Table I
INDICATIONS FOR HYSTEROSTOMATOMY

Fetal Indications	
Fetal distress	27
Prolapsed cord	3
Transverse presentation	1
Postmaturity	2

Maternal Indications	Combined	Alone
Exhaustion	28	1
Occiput posterior	23	6
Transverse arrest	16	0
Bandl's contraction ring	1	0
Uterine inertia	5	1
Elderly primipara	1	0
Pre-eclampsia	7	1
Chronic nephritis	3	0
Previous cesarean section	1	0
Disproportion	5	0
Face presentation	2	0
Brow presentation	1	2
Cervical dystocia	19	2
Breech	3	1
No progress	1	0
Eclampsia	1	0
Premature separation of placenta	1	0

Labor and Presentation. In fifty-one cases where prolonged labor was one of the indications for delivery the average duration was 41.8 hours. In sixty cases the presentations were other than occiput anterior; thirty were occiput posterior; twenty-one were transverse arrest; two were face; two brow; one transverse presentation; and four breeches.

Indications. In most instances the indications for the operation were multiple and included both the mother and baby. (Table I.)

Methods of Delivery. The methods of delivery (Table II) give some idea as to the types of cases. In sixty-nine cases (primigravidae) episiotomy was necessary.

Table II
METHODS OF DELIVERY

Low forceps	20
Mid forceps	31
Mid axis traction	12
Scanzoni	3
Breech extraction	4
Internal version and extraction	5
Craniotomy	3

Types of Incisions and Extensions. The usual incisions in the cervix are generally made at 10, 2, and 6 o'clock but in this series we find that in fifty cases the incisions were made at 10 and 2; in seventeen cases, at 10, 2, 6; in four cases, only one incision; and in eight cases the number of incisions was not noted. It has been an interesting finding that in the majority of the cases the posterior lip of the cervix is fairly well thinned out while the anterior lip remains rather thick, so that only the 10 and 2 o'clock incisions seem to be necessary. In nine cases the incisions in the cervix extended and lacerations occurred in addition to the incisions. It is important to note that these lacerations and extensions were noted only in those cases where but two incisions into the cervix were made. This would indicate that it is definitely safer to make the three incisions in order to avoid extensions or lacerations.

Morbidity. It is to be expected that in cases in which there are generally multiple indications for delivery, where the mother is exhausted by long labor, and where surgical interference becomes necessary, morbidity would be high. In this series the morbidity was 24.36 per cent.

Fetal and Maternal Mortality. Twenty-one babies died: fifteen were full term; two premature; and four were dead before delivery, Thus giving a gross fetal mortal-

ity of 21 per cent. The maternal mortality in this series was one, or 1.28 per cent.

Weights of Babies. Weights of infants are given in Table III, with indication of prematurity, maturity and postmaturity. There was a high incidence of large babies, 65 per cent being in the group from 3000 to over 4000 gm.

TABLE III
WEIGHTS OF BABIES

Gm.	
Less than 1500	1
1500–2500	6
2500*–3000	21
3000–4000	45
Over 4000	7
Total	80

* Two sets of twins.

Postpartum Hemorrhage. Postpartum hemorrhage was increased in this series. No exact method of measuring the amount of blood loss was employed and only approximate estimations were made. Considering an estimated loss of 500 or more c.c. as a postpartum hemorrhage we find that this occurred in eleven cases, or 12.8 per cent.

Healing of the Cervix. We were very much interested in following these cases to learn the condition of the cervix at the six weeks postpartum examination and we were successful in securing data on this question in forty-eight patients. In forty-two of the patients the cervix was considered as well healed; in five as poorly healed; and in one case the cervix was adherent to the lateral walls of the vagina.

Subsequent Pregnancies. We also were interested in the possible effects of hysterostomatomy on subsequent pregnancies as well as possible sterility. Since our series has been compiled in the last three years it is not expected that we would have sufficient data to answer these questions. However, we were able to find that in nine patients subsequent pregnancies have occurred. Seven of these have already been delivered of full term babies. Two labors ended spontaneously, three were assisted with prophylactic low forceps, and two required cesarean section. Of the two cesarean sections, one was done for cephalopelvic disproportion as an elective procedure; and the other, after a trial labor with failure of the cervix to dilate, was done by low cervical section. In the five cases that were delivered per vaginam the shortest labor was two hours and forty minutes, the longest, eleven hours and thirty-eight minutes. The average duration of labor was six hours and eight minutes. In none of these five deliveries were incisions of the cervix necessary and in three cases inspection of the cervix following delivery showed lacerations. The weight of the babies delivered ranged between 3,515 and 4,649 gm.

CONCLUSIONS

1. Hysterostomatomy (Dührssen's incision) is a valuable procedure in obstetrics.
2. The indications for this procedure are generally multiple, involving both mother and child.
3. The 10-2-6 o'clock incisions will prevent extensions and lacerations of the cervix.
4. The maternal morbidity and fetal mortality are necessarily increased.
5. The babies in these cases have a tendency to be large and overweight.
6. The cervix, in the great majority of cases, heals satisfactorily.
7. Postpartum hemorrhage is increased. This can be attributed to the general exhaustion of the patient and to the operative procedures necessary to complete delivery.
8. There is no tendency to sterility following operation.
9. Subsequent labors are not necessarily prolonged or interfered with.

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SOME COMPLICATIONS OF A COLOSTOMY*

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TO perform satisfactorily and be of least annoyance to the individual, a colostomy must be so constructed that it will discharge the fecal content of the bowel completely and with minimum danger of later complications. An improperly made artificial anus will not only be an annoyance to the patient, but will lend itself to accidents, that if foreseen, could in many instances have been avoided. Nearly all complications can be prevented if due attention is given to planning of the operation and time given to instruction as to the care of the colostomy in the daily cleansing. The position of the opening has much to do with the function of the bowel; whether the opening is in the portion of the gut containing semi-liquid feces or in a portion of the bowel with solid feces, will determine to a large extent how it will operate, and how it works will determine largely the comfort or discomfort of the patient.

One major complication that should be charged to a poorly constructed colostomy or an opening in a patient poorly instructed in its care, is the mental distress that may progress to the point where the individual shuns contact with society and becomes unfit to carry on his ordinary business. This need not be, for in a properly constructed and properly managed colostomy the man is able to carry on his work, laborious though it may be, with comfort and a minimum amount of care.

How soon a colostomy should be performed has been a point for argument over long years and while it is still a subject of discussion, the tendency now is to make a colostomy early. This holds good particularly for the inoperable cases of cancer of the rectum; to withhold a colostomy until ulceration about the cancer has passed beyond the wall of the bowels is apt to make of the colostomy only one more complicating factor in the progress of the disease.

In the operable cancer of the rectum, the time of making a colostomy may to a certain extent be predicated upon the location of the growth and upon the time that resection is expected to be done. At the rectosigmoid a cancer can be expected to produce obstruction sooner than an involvement of the ampulla. Because of the capaciousness of the ampulla, obstruction may never occur, but at the rectosigmoid it may appear abruptly without much warning. A colostomy in the presence of obstruction, becomes a complicated procedure with a rapidly rising death rate.

In the inoperable carcinoma no subsidence of the disease can be expected, so that if any benefit is to be derived from the colostomy, it should be made early. It seems reasonable to consider the making of an artificial opening as soon as the diagnosis of inoperable carcinoma is made. Miles[1] sums this up very well when he states that as soon as carcinoma of the rectum is found to be inoperable, every day lost before resorting to colostomy is a day to the bad.

The type of colostomy will have something to do with the complications and accidents that follow. A simple loop colostomy, in which there is not a good spur, will give trouble because of the spilling over of feces into the distal limb, which becomes not only an annoyance to the patient, but a danger as well. Where the gut is divided, the ends widely separated and the abdominal wall closed between them, one may expect a colostomy to function to the

* Read before the Proctologic Society of the Graduate Hospital, University of Pennsylvania, February 8, 1939.

highest point of efficiency, because all feces will discharge directly to the surface and only a small portion of the gut is exposed. This type of colostomy will require somewhat longer to construct and, if care is not exercised, may show a higher mortality.

The complications that arise from the location of the artificial opening on the abdomen are largely due to the inability of the patient to give proper attention to the opening. Improper care or lack of care invites the irregular action of the colon with its soiling, and the mental distress due to a sense of insecurity.

Peritonitis is an ever present menace to surgery of the colon, particularly in those cases of malignancy where extensive involvement has occurred. It is easy, with injudicious handling of a growth, to express infectious material through the damaged bowel wall. The incidence of peritonitis is remarkably higher in this type of surgery than in gastric or pelvic surgery, because of the anatomic conformation of the large bowel and its normal bacterial contents.[2]

Obstruction caused by the passing of the small intestine around the attachment of the sigmoid to the abdominal wall when care has not been used in closing the space between the colon and the lateral abdominal wall, is a complication that can be avoided by careful technique when making the colostomy. This technique should in some way completely obliterate the space between the sigmoid and lateral wall, at the same time allowing the knuckle of gut to come out through the abdominal opening with good spur formation, if a loop colostomy is made. If obstruction does occur it becomes a serious accident with a high mortality.

Various types of hernia may occur through the opening made in the abdominal wall to accommodate the protruding gut. The simpler type shows gradual dilatation of the abdominal opening with formation of globular distention under the skin, pushing the posterior wall of the protruding gut forward. This forward protrusion of the colostomy becomes a nuisance to the patient; it is sometimes visible under the clothing; and it is always productive of increased moisture and because the surface is subject to increased traumatism, there may be bleeding. The wearing of bags with hard ring edges at the edges of receptacles will produce this type of hernia.

Pemberton[3] reports an unusual complication in a man with a colostomy who returned in one year because of hernia of the abdominal incision with prolapse of both limbs of the loop. This was repaired. In a little more than a year he returned because of another prolapse larger than he had ever noticed before. Attempts at reduction resulted in perforation of the prolapsed portion of the colon through which 2 feet of small intestine protruded.

Separation of the wound edges is not unusual in patients suffering from malignancy, and that being so, rupture of the wound in colostomy need not be an unexpected accident.

A man was admitted to the Atlantic City Hospital with a carcinoma of the rectum. A colostomy was done, with separation of the ends of the bowel and repair of the abdominal wall between. Two weeks after operation, during a severe fit of coughing, the wound opened and a loop of small intestine protruded between the ends of the colostomy. This accident is not an infrequent occurrence. In poorly nourished individuals the danger should be anticipated and steps taken to support the wound. When it does occur the protruding intestines are always soiled by extruded feces. However, because of increased tolerance due to autovaccination, peritonitis is less frequent than might be supposed.

An annoying complication is prolapse of the colon through the proximal opening which may under some circumstances result in protrusion of a large section of the gut. While several factors enter into the production of a prolapse through the proximal opening, the most common cause is to allow too much sag or freedom of movement in the proximal loop. This has often been presented as an argument against the formation of a colostomy.

It is a well known fact that scar tissue in the skin will contract more than in other organs, and therefore, if care is not taken in closing the skin around the gut, stenosis may occur. Some surgeons, to obviate this difficulty, remove an elliptical section as a preliminary to the abdominal incision. When stenosis does occur it frequently requires some form of plastic operation to secure a properly functioning outlet. The stenosis of colostomy opening will be seen more often around a single barreled colostomy than in a loop colostomy, since often a stab wound is made with tight fit around protruding gut.

We have had the unfortunate experience of extensive infection around the artificial opening and attributed the invasion to the fact that the bowel was sutured to the abdominal wall. We no longer anchor the bowel in any way that may require introduction of sutures into its walls.

In the care of a colostomy, we carefully instruct the patient in the necessity of properly irrigating the proximal colon at regular intervals. Caution should always be used in introducing a rectal tube into the colon; no force should be applied to the advancing tube and a large, stiff piece of rubber should not be used. If caution is not exercised the tube may puncture the bowel even after long familiarity with its use. In one of our patients, a man who for five years satisfactorily irrigated the proximal portion of the gut, this accident happened. One morning, due to lack of time, he applied undue force to the entering tube which penetrated the colon, and he then proceeded to inject at least a pint of water into his abdominal cavity.

Extension of the original disease into the colostomy is not an infrequent occurrence and can, to a certain extent, be avoided by being sure that the portion of the gut selected for the opening is free of disease, is a sufficient distance above the lesion, and that a similar invasion has not occurred about the point selected for section.

Bleeding from the protruding mucosa usually alarms the patient, but this ordinarily is caused by trauma of the material covering the exposed mucous membrane and is usually rectified by attention to detail in the application of the dressing.

There is a very serious complication that may arise as the result of division of the mesentery in the formation of a single opening. The blood supply of the left colon is not so evenly distributed as that of other portions of the colon, so that if vascularity of the bowel is not certain, necrosis of the gut may occur with rapid onset of peritonitis.

Parotitis is frequently seen where the parotid gland lacks the stimulus of food taken by mouth. It may occur in various degrees of severity, often to the point of suppuration. It is well to have these patients chew gum or suck upon pieces of orange or lemon.

SUMMARY

To fulfill its purpose, a colostomy must function without distress or discomfort and with a minimum amount of attention.

There should be no mental hazard associated with an artificial intestinal opening, as otherwise a large proportion of the benefit is lost.

Many complications that arise as the result of the operative work or in the care of the colostomy, should be avoided by careful planning of the operative technique and a clear understanding of what is to be expected of the abdominal anus.

It is necessary to avoid or lessen the possibility of subsequent accidents if we expect to offer the colostomy as a substitute for a normal anus.

A colostomy should be so devised and constructed, and its after-care such that its presence will be only a minor handicap.

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TUMORS ORIGINATING IN THE ISCHIOANAL FOSSAE

REPORT OF CASES

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ALTHOUGH tumors, exclusive of inflammatory processes, originating in the ischioanal fossae are uncommon, a particular problem in diagnosis and treatment arises when the condition is encountered.

In 1908, Chavelet was able to collect only eleven reports of neoplasms in this location, three of which were myomas, three fibromyomas, one a lipoma, one fibroma, one fibrolipoma, and two sarcomas. The only other report that I was able to find in the literature was one by Shedden in 1934, in which he reported three cases of sarcoma arising in the ischioanal region.

This study is based on a series of four cases seen recently at The Mayo Clinic.

As an aid in studying these reports and in understanding the path followed by the tumor as it enlarged, a review of the anatomy is indicated. In this study the term "ischioanal fossa" will be used instead of "ischiorectal fossa" because the levator muscle is attached to the wall of the anal canal rather than to the wall of the rectum, and this fossa actually bears no immediate relation to the rectum but rather to the anal canal—hence the adjective "ischioanal."[1]

The ischioanal fossae are situated on either side of the anus and are normally filled with fibrofatty tissue. Each is pyramidal in shape, with the base pointing backward and the apex anteriorly and medially. Anteriorly they are separated from each other by the rectum and prostate (or vagina). Posteriorly they are in contact except at the anococcygeal raphé. Their lateral walls are relatively rigid and are formed by the ischial ramus, covered by the obturator internus muscle and aponeurosis. Lying in a reduplication of this aponeurosis are the internal pudic vessels and nerves. The medial wall is relatively mobile and is made up from above downward of the levator ani, internal sphincter and external sphincter muscles. The levator ani muscle separates the fossae from the subperitoneal or pararectal space. The bases of the fossae are represented by the superficial fascia and skin. The apex is formed by the junction of the levator fascia and that of the obturator internus. The fossae measure approximately 5 cm. in the anteroposterior diameter, 2.5 cm. laterally, and 5 cm. to 7 cm. vertically. The fossae are lined with fascia covering the superficial surfaces of the obturator internus and levator ani muscles. This fascia is continuous with the fascia covering the gluteus maximus muscle, laterally, and Colles' fascia, anteriorly. The inferior hemorrhoidal vessels and nerves pass through the ischioanal fossa. Posteriorly, between the two fossae, is a fat-filled space by means of which the fossae can communicate by rupturing a very thin fascial layer.

It is therefore conceivable that new growths in this region could be either mesodermal or ectodermal in origin.

REPORT OF CASES

CASE 1. A farmer, male, aged 49 years, registered at The Mayo Clinic because of a painless swelling in the region to the left of the anus, which he had noticed for about three months. Because of the progressive enlargement, it was causing him inconvenience on sitting or riding farm machinery. The results of proctoscopic examination were negative. Palpation disclosed that there was a circumscribed, movable tumor approximately the size of a baseball in the region of the left ischioanal fossa

which was not tender and to which the overlying skin was nonadherent. The mass was completely removed and it was found to be well encapsulated except at one point where it was attached by a pedicle in the region of the ischial ramus. Examination of the tumor revealed that it measured 7 cm. by 6 cm. by 5 cm. The microscopic report was "lipomyxoma containing small areas of bone." Recovery was uneventful and the wound healed by primary intention.

Case II. A farmer, male, aged 50 years, came to The Mayo Clinic because of a painless tumor in the region of the left ischioanal fossa which, because of its size, caused some difficulty in sitting, and because of its location, caused difficulty in the local hygienic care of the anus. The tumor was found to be movable and well circumscribed. It was excised and proved to be about 10 cm. by 9 cm. by 8 cm. in size. The microscopic report was "lipoma." Recovery was uneventful.

Case III. A man, aged 64 years had noticed a painless, progressively enlarged swelling to the right of the anus for one year and this was a source of concern because of his fear of cancer and the difficulty he encountered in the care of the anus after defecation. On digital examination of the rectum, the tumor could be palpated as an extrarectal mass on the right side. It seemed to be adherent to the wall of the rectum. It was excised and was found to be adherent to the levator ani muscle and wall of the rectum. The tumor was 10 cm. by 10 cm. by 10 cm. in size. The microscopic report was "fibroma, probably originating as a lipoma." Recovery was uneventful.

Case IV. A farmer, aged 28 years, was brought to The Mayo Clinic because of swelling and tenderness to the left of the anus for ten days. On examination, a tender, bulging mass with reddened, overlying skin was discovered. Clinically, it was thought that this was an ischioanal abscess, and incision for drainage was attempted. However, it was found to contain hair and the usual necrotic débris of a dermoid. After the acute inflammatory process had subsided, the cyst was removed. The microscopic report was "squamous epithelial lined cyst."

Tumors of the ischioanal fossa, if nonmalignant or noninflammatory, are usually painless and give rise to symptoms such as discomfort in sitting and with bowel movement, only because of their size and location. They vary in size, and a combined rectal and vaginal examination may be of value in determining the size, degree of mobility and point of origin. Pressure on the contiguous structures may cause pain or alteration of such physiologic processes as defecation or urination. Unless secondarily infected, the overlying skin is freely movable.

These lesions must be differentiated from such pathologic processes as ischiorectal abscesses, pilonidal disease, chronic anal abscesses and perineal hernias. Ischioanal abscess is the most common lesion occurring in the region of the ischioanal fossa. It can be differentiated from benign tumors by the speed of development, fever, leucocytosis, tenderness, fluctuation and pain. It is more difficult to rule out an abscess of the lumbar region, such as occurs in Pott's disease, which may point near this region. However, the course of the disease and roentgenologic examination of the spine would be of help in diagnosis.

Pilonidal cyst disease, although usually located in the coccygeal region, may extend anteriorly to the midline of the anus or into the perineum. Perineal hernias are very rare, but can usually be differentiated by reducibility.

Chronic ischioanal abscesses or "incomplete" external fistulas usually are surrounded by a firm, fibrous or scarred wall and may be very difficult to differentiate from the rare tumors. Careful examination of the dentate margin to discover the primary source of the abscess in an anal crypt and palpation of the anus and perianal tissues between the index finger and thumb usually will disclose the site of origin.

SUMMARY

Tumors originating in the ischioanal fossa are rare. Such tumors present problems interesting in differential diagnosis and treatment.

The subjective symptoms caused by tumors of the ischioanal fossa, as shown by these cases, are not definitely peculiar to the lesion but in general include painless, progressive swelling which causes difficulty on sitting or defecation. The findings of asymmetry in the ischioanal region, overlying normal skin, and circumscribed movable tumor mass that is not tender are of value in differential diagnosis.

These tumors should always be excised and the tissue examined microscopically for the correct diagnosis.

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In about 80 per cent of cases, carcinoma of the stomach occurs in a patient who has never been previously sick. Not infrequently he volunteers the information that it is the first time in his life that he has consulted a doctor.

From—"Surgery of the Alimentary Tract" by Devine (Williams & Wilkins Company).

OXYGEN VENTILATION IN THE TREATMENT OF BLADDER TUBERCULOSIS

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TUBERCULOSIS of the bladder is, essentially, a secondary lesion dependent upon a focus elsewhere in the genitourinary tract, usually the kidney or prostate. Primary tuberculosis of the bladder is such a rarity that its existence is open to question and, for practical purposes, one is justified in considering each case as a secondary lesion. Symptoms in fully 60 per cent of the cases promptly abate following extirpation of a unilateral tuberculous kidney. Local treatment, therefore, is withheld for a time after nephrectomy, but is indicated should improvement fail to occur after a period of six months. Subsidence of bladder symptoms is more likely and more prompt when the preoperative symptoms are of short duration. Even after unilateral nephrectomy a small percentage of patients will have persistent bladder symptoms resisting all forms of therapy, local and general, which are currently employed.

Source of Infection. Infection may reach the kidney by way of the blood stream, lymphatics, by extension through the ureter and by direct extension. The hematogenous route is most plausible. Invariably the primary renal focus is situated at the base of the pyramid in the region of the medullary portion of the renal cortex. Tuberculous pyelitis with its attendant symptoms develops when the process extends in all directions and breaks down spontaneously into the renal pelvis which is near the cortical surface.

Pathology. Tubercles, both miliary and conglomerate, occur in the mucosa, especially the trigonum, followed later by shallow, ragged ulcerations with nodular bases. Halle and Mortz divided the progress of vesical tuberculosis into three stages: the first including tubercle formation and superficial ulceration preceded by an edema and reddening in the region of the ureteral orifice; the second being the stage of deep ulceration involving the muscular layer; and the third the destructive stage.

The tubercle bacillus is an aerobe and will not grow under strictly anaerobic conditions. Novy and Soule (1925) found that the optimum oxygen pressure for growths of the human type of organism was 40 to 50 per cent, while above and below this pressure growth was retarded. Carbon dioxide seemed to have little effect on growth; no inhibitions occurred till the concentration reached 60 per cent, and even in 90 per cent carbon dioxide fair growth occurred.

Symptoms. The symptom-complex of tuberculosis of the bladder is essentially that of renal tuberculosis. The renal lesion or primary focus is frequently suggested by the evidence of tuberculous cystitis. Frequency of urination, dysuria, and varying degrees of hematuria form the chief symptoms. The initial symptom of frequency varies in intensity and severity, and with exacerbations and remissions, tends to become chronic. Dysuria varies with the extent of the bladder ulcerations and may amount to extreme tenesmus. Hematuria is most variable in degree and occasionally may be massive. Systemic symptoms as vesperal fever, loss of weight, night sweats, pulmonic tuberculosis, etc., are often present.

The persistence of bladder symptoms following nephrectomy, according to Cabot, may be due to one of four reasons: (1) severe invasive cystitis with secondary contracture; (2) unsuspected dormant le-

sion in the remaining kidney; (3) pronounced vesical ulceration; (4) contracture of the vesical neck.

In a large percentage of cases even ulcerations do not prevent subsidence without supplementary treatment. Even contractures in themselves will improve through distention, either natural or artificial hydraulic.

Diagnosis. A diagnosis of tuberculous cystitis is frequently rather taxing. A history with especial reference to the triad of frequency, dysuria, and varying hematuria is of utmost importance. A thorough physical examination for possible primary focus of infection elsewhere in the body is necessary. Laboratory work is concerned chiefly with urinalysis and guinea-pig inoculation. A so-called "organism-free pus" is presumptive evidence of tuberculous cystitis. The presence of the tubercle bacillus in urine is an absolute assurance of tuberculous infection in the urinary tract. This is easily proved by the carbol-fuchsin stain of the urinary sediment. Guinea-pig inoculation tests are positive. Cystoscopy will reveal tubercle formation and ulceration in the region of the ureteral orifice and trigonum.

A plain *x*-ray plate of the abdomen will often show tuberculous calcareous deposits irregular in shape and density. A cystogram may outline a bladder contracted more in one-half. A marked dilatation of the bladder sphincter and posterior urethra in the presence of a severe cystitis is highly suggestive.

Treatment. The local treatment of tuberculous cystitis is diverse. Thus, Herman recommends instillations of ½ ounce of gomenal in oil, 10 to 20 per cent, three times weekly, or 10 c.c. of a mixture of calomel 2, guaiacol 15, and sterile olive oil 100, once or twice weekly. Casper recommends the instillation of 20 to 30 c.c. of a 1:20,000 solution of bichloride of mercury, gradually increasing the strength to a 1:2000 solution. Hollander prescribes potassium iodide orally and a calomel emulsion instilled into the bladder to liberate mercuric iodide. Silver nitrate 1:1000 is useful only in cases with a mixed infection, and is intensely irritating in the presence of open ulcers. Greenburg and Brodney advised methylene blue in 2 gr. capsules three times a day and an instillation of a 1 per cent c.p. solution into the bladder. Rousing suggested the use of phenol ½ to 6 per cent as a bladder instillation. Iodoform (5 to 10 per cent) or guaiacol 5 per cent in liquid petrolatum, picric acid 0.5 to 1.0 per cent, and mecurochrome 1 per cent, have also been used. Santol oil 5 to 10 minims and hyoscyamus 10 to 60 minims are useful internal remedies. Myll states that symptomatic improvement follows the intraveous injection of 5 c.c. of a 5 per cent solution of calcium chloride. The injections are made at five day intervals. Wizack employed lactic acid instillations in strengths of 20 per cent with satisfactory results. Caulk and Ewerhardt have used intravesical ultraviolet medication and air ventilation with beneficial results.

Various surgical procedures such as suprapubic cystostomy, ureterostomy, uretero-intestinal anastomosis and fulguration of the bladder mucosa have likewise been advocated from time to time.

In rebellious cystitis cases which occasionally follow nephrectomy and which resist all standard forms of medication I have used pure oxygen ventilation and irrigation of the bladder with gratifying results. I have been unable to find any report of this therapy being used elsewhere.

Technique. The bladder is completely emptied by means of a catheter and repeatedly irrigated with pure oxygen. Finally, with the bladder under slight oxygen distention, the catheter is removed. These oxygen irrigations are given at weekly intervals. During this course of therapy no germicidal solutions are used. Cystoscopic bladder examinations are done at regular intervals.

Results. Although the number of tuberculous cystitis cases seen at the Church Home and Infirmary is limited, I have had

gratifying results in two of our most resistant cases.

Case 1. M. T. age 17 years, was first seen on February 16, 1937, there was a history of frequency, nocturia, dysuria, and hematuria of two months' duration. Kelly air cystoscopy revealed a severe hemorrhagic cystitis with hemorrhagic areas and diffuse tubercles scattered over the trigonum and posterior bladder wall and in the region of the right ureteral orifice. The urine was loaded with pus cells, but no organisms could be seen. Bladder culture was sterile. Repeated bladder specimens were negative for acid-fast bacilli. Guinea-pig inoculation was positive for tuberculosis.

On March 3 *x*-rays were taken. Numerous calcified bodies were seen in the region of the right kidney and the pyelogram suggested acid-fast infection due to the "fuzzy" appearance of the right ureter. Sterile cultures were obtained from both kidneys. Urinary sediment examination from the right kidney was positive for acid-fast bacilli. Guinea-pig inoculation of urine from both kidneys was done, with the right proving positive and the left negative. Chest *x*-ray was negative for tuberculosis. The Wassermann and Kahn tests were negative.

On June 9, 1937 a right nephrectomy and partial ureterectomy were done. The immediate postoperative course was uneventful. Pathologic examination showed tuberculosis with cavitation at the lower pole of the right kidney, tuberculous pyelitis, and ureteritis.

The patient was discharged on June 20, 1937, but her bladder symptoms were only temporarily relieved. She was not treated locally.

In December, 1937 she complained of an exacerbation of the urinary symptoms, the frequency, dysuria, and hematuria being more pronounced. Cystoscopy showed marked ulceration and tubercle formation involving the right half of the bladder. Weekly bladder instillations of 10 per cent argyrol or gomenol in oil produced no relief. On February 11, 1938 urinary sediment from the bladder showed numerous acid-fast organisms present. Throughout the next two months the patient complained of increasing severity in her urinary symptoms. Since all of our standard treatments were of no avail, on March 3, 1938 the first treatment of oxygen ventilation was carried out and others were continued at five day intervals. On March 28, 1938 the patient stated that her symptoms had subsided tremendously. Repeated smears of the urinary sediment showed a marked decrease in the number of acid-fast bacilli seen. Guinea-pig inoculation of urine from the left kidney was negative. Cystoscopic examination of the bladder on April 21, 1938 showed only a small area, 0.5 cm. in diameter, still ulcerated. The previously noted ulceration and tubercle formation had subsided. Oxygen instillations were now given at weekly intervals. On April 27, 1938 the bladder urine sediment had only one acid-fast organism after extensive examination. Cystoscopy at this time showed the bladder ulceration entirely healed. On May 4 and May 10, the urinary sediment was examined and no acid-fast bacilli were seen. This patient is now being watched without treatment and is entirely asymptomatic.

Case 11. E. K., age 50 years, had had a right nephro-ureterectomy in 1934 for tuberculous ureteritis and pyelonephritis of three years' duration. This was followed by a rebellious cystitis which had been treated by the usual standard methods but without results. Nocturia, dysuria, and diuria were marked. Hematuria was variable but never massive. Urinary sediment stains showed the presence of acid-fast bacilli. Cystoscopy revealed that the right side of the bladder mucosa and trigonum were replaced by large ulcerations and conglomerate tubercles. A guinea-pig inoculation from the left kidney was negative. The bladder capacity was 300 c.c.

In September, 1937 oxygen ventilation was started at weekly intervals. In four weeks there was a marked subsidence in the urinary symptoms. Cystoscopy was done in January, 1938 at which time the ulcerations noted previously were healed but the tubercle formation persisted. The oxygen ventilation was carried out until March. There has been no recurrence of the symptoms although tubercle bacilli may be found in the bladder urinary sediment after careful searching. This patient was asymptomatic for six months.

CONCLUSION

The various forms of therapy offered for rebellious tuberculous cystitis following nephrectomy are so diverse that no one treatment is successful in all cases. I have used pure oxygen ventilation and irrigation of the bladder with good results in two cases. Believing this may offer an

adjunct in the palliative treatment of vesical tuberculosis this résumé is presented as a preliminary report until the merits of the method can be determined.

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Tuberculosis of the arteries is the result of direct extension of ulcerative lesions in the lungs or the breaking down of tubercular glands in the chest.

From—"Peripheral Vascular Diseases" by Kramer (Blakiston).

A CONSIDERATION OF THE SUTURE PROBLEM*

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THE existence of the suture problem is manifested by the variety of suture materials at hand as well as by the lack of uniformity in the methods of their utilization. Yet, the suture and its application is such an integral part of the surgeon's, and for that matter, the general practitioner's daily activities that it is too often taken for granted. Most of us, unfortunately, acquire our suture technique rather blindly from our elders and with it many time-honored and often time-disproved prejudices.

At present, the ideal suture material does not exist, and the materials available represent compromises. The absorbable sutures produce varying degrees of tissue reaction, and their rates of loss of strength and of absorption are far from constant. Some of the non-absorbable sutures are remarkably inert in tissue and they maintain tensile strength far beyond any necessary time interval, but, as the name implies, are of the nature of permanent guests. It is not too much to expect that the future will produce a material with the advantages of both and the disadvantages of neither. In the meantime, a knowledge of the characteristics of both types is of considerable advantage to the surgeon.

ABSORBABLE SUTURES

Absorbable sutures are now made exclusively from animal tissues. Catgut, kangaroo tendon and preserved fascia comprise almost the entire group, although in Germany "carnofil" made from serum has had some clinical acceptance. Catgut is far and away the predominant material, and is obtainable as "plain," chromicized, iodized, tanned, or processed by combinations of these methods. Huhne, in Germany, has introduced another form of processing involving certain aniline dyes, but this catgut is not available in America at present. According to its discoverer, it eliminates most of the disadvantages inherent in the use of catgut. Some of Huhne's claims have been corroborated by other observers.

Catgut's sole advantage lies in its absorbability in the body tissues, which is necessarily associated with a progressive loss of tensile strength. Chromicizing and the other processes are used in an attempt to control this loss of strength. Plain catgut loses about 90 per cent of its strength within six days, and the processed catguts within ten to twenty days, under ideal wound conditions. Many investigators, including Howes and Harvey, Jenkins, Kraissl and Inman, have shown conclusively that the optimum ten, twenty and forty day absorption rates claimed by manufacturers are subject to wide variations under clinical as well as experimental conditions. Inman, in this hospital, demonstrated that plain catgut, when placed subcutaneously in clean herniorraphy wounds, lost most of its strength within three to six days; forty day chromic 2 catgut similarly placed was without measurable strength within ten to fifteen days, and frequently as early as the seventh day. This is in agreement with Jenkins' work. The latter, however, found one type of chronic gut which seemed to approximate the manufacturer's claims.

It is generally recognized that wound infection markedly accelerates the rate of digestion of all types of catgut and there is some reason to believe, as does Hinton,

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that certain individuals are allergic to catgut and manifest this allergy by accelerated suture digestion. Clinically, the disappearance of absorbable sutures in many cases of wound disruption gives credence to this viewpoint, although the presence of subclinical infection may also be a factor. There is a suggestion in the work of Ravdin and his collaborators that protein deficiency may influence the rate of catgut digestion. The experimental and clinical studies of the increment of strength in the healing wound together with the studies of the rate of absorption of catgut, however, give sufficient information to allow the rational use of catgut under average circumstances.

Aside from inconstancy of loss of tensile strength there are several other factors which make catgut a less than ideal material. A definite foreign body reaction is seen surrounding the strands, which is more evident when the more durable, chromicized varieties are used. This is expressed clinically by the increased induration about the wound in which catgut has been used. Another disadvantage is the necessary reliance upon the manufacturer for the control of sterility. Meleney and Chatfield, Clock and the British commission have shown that catgut purchased in the open market is not invariably sterile when subjected to exhaustive test. It must be said, however, that the major American manufacturers have adopted the rigorous standards and testing techniques of Meleney and of Clock, so that the possibility of purchasing contaminated catgut in the domestic market is practically nil.

NON-ABSORBABLE SUTURES

There are almost as many varieties of non-absorbable sutures as there are raw materials available for their fabrication. To mention a few: silk, linen, cotton, stainless steel, silver, bronze, horsehair and silkworm gut may be purchased in the average surgical supply house and are in use in one or more clinics throughout the world. Among the metals, stainless steel and silver are most frequently used. The other metals are used occasionally, but possess the disadvantage of causing tissue necrosis in varying degree because of chemical interaction with the tissues. In this connection it is important to realize that the term "stainless steel" includes a wide variety of alloys, many of which definitely interfere with healing. The alloy recommended by Babcock has been tested extensively and shown to be inert in tissue.

Silk is the favorite material among the non-metallic sutures, combining as it does high tensile strength, pliability and lack of tissue reaction. Many surgeons have great fear of infection in its use, but it is more sinned against than sinning. Halsted had little fear that silk, properly used, would contribute to infection. Shambaugh, reporting the results of the use of silk and catgut in herniorraphy over a twenty-one year period, found that infection was less frequent when silk was used, and further that infection persisted longer in the catgut series. In this study the nature of the suture material was the only variable factor.

Horsehair is frequently used in facial surgery because it can be obtained in very small diameters. It has little use elsewhere and, even in the face, may be replaced with silk.

Silkworm gut is mentioned only to condemn it. Surgical silkworm is a very poor relation of the elegant gut used for leaders, and is brittle, hard to tie securely, and seems to be irritating. Its advantage, lack of capillary action, is possessed by the metallic sutures and by processed silk and linen. Every one who has used it for tension sutures has had the discomforting experience of finding one or more silkworms broken at the time of the first wound inspection. However, its use is deep-seated in tradition and will probably continue for many years.

PRACTICAL CONSIDERATIONS

Hemostasis. As the primary purpose of any material used for hemostasis is the

closure of the vessel involved, the size of the suture required is dependent on the nature and caliber of the vessel. Either absorbable or non-absorbable sutures may be used in the non-infected wound, but catgut is usually chosen in the presence of infection. Kennedy, however, uses silk exclusively regardless of the nature of the wound.

The common error in the selection of "ties" is the use of unnecessarily heavy material. This fault is largely confined to the "catgut" school, and the influence of the silk advocates is perhaps chiefly responsible for the trend toward smaller sizes in absorbable material. Despite this, one frequently sees vessels tied off with sutures whose diameters are larger than the vessels. With the exception of major arteries, any vessel can be securely fastened with oo catgut, and even smaller sizes are adequate for most "bleeders." Those who are accustomed to the larger sizes complain that the smaller ties break with great frequency and the complaint is not entirely unreasonable. The answer lies in the manner of tying and in the amount of tissue included within the tie. If the vessel alone is included and the knot made without terrific tension there will be no trouble. Too often we forget that the tension within the included tissue is governed by the tension of the first throw, and that the second throw merely acts as a lock for the first. Observation will reveal that most sutures are broken in an attempt to put unnecessary tension on the second knot. If extra security is needed, a transfixion suture will be adequate.

Wound Closure. The term covers a tremendous variety of situations, but intestinal suture, abdominal wall closure and skin suture will serve to illustrate the basic problems. After all, sutures are employed in any wound solely as an artificial support until the tissue union has attained sufficient tensile strength for normal activity. Consequently, the requirements for suture tensile strength and durability are governed by the tissues and the forces which tend to distract them.

The suture par excellence for intestinal work is silk, as Harvey has recently pointed out. The fine interrupted suture provides a maximum of strength, and a minimum interference with healing. If catgut is to be used, ooo is large enough and Bower's work indicates that ooooo will give even better results. In my experience a two layer suture is adequate, when interrupted black silk seromuscular sutures are used. The inner mucosal suture may be carried out with a fine running catgut stitch.

The conventional abdominal wall closure is carried out by suture of the individual anatomic layers, although Kennedy and more recently Hinton (upper abdominal incisions) advocate the "through-and-through" one layer closure. This type was abandoned generally after Billroth demonstrated the markedly lessened incidence of postoperative hernia following anatomic reconstruction. Kennedy reports a gratifyingly low incidence of wound disruption and hernia following the single layer closure. It is highly probable that complications as a result of "through-and-through" closure are less frequent than in Billroth's era, but closure layer by layer appeals to the vast majority of surgeons.

If the wound is likely to be infected, as after the incision of an abscess, or generalized peritonitis, or if there has been gross contamination intra operationem, it is advisable to use absorbable sutures. For clean wounds absorbable or non-absorbable sutures may be used at the option of the surgeon. In either case, it is most important to recognize that the only layers with appreciable tensile strength are the fascial sheaths and aponeuroses. Double zero plain catgut or very fine silk is strong enough if the peritoneum is closed as a separate layer. If the posterior rectus sheath is included, o chromic or silk with the equivalent tensile strength is adequate. During the past three years, I have used oo chromic without mishap. We have demonstrated that the fascia of the anterior rectus sheath, when sutured with o chromic gut and placed in a tension

dynamometer, tears beyond the suture line before suture breakage occurs. If muscles are to be approximated, very fine sutures are advisable because muscle will tear long before the breaking point of the suture is reached.

Skin closure may be carried out with any fine, non-irritant material.

While the decision for or against silk is best left in the hands of the individual operator, I have been impressed by the lack of induration in the wound sutured with silk or stainless steel and, as a result, have largely abandoned the use of catgut.

The custom of using so-called tension sutures to relieve strain on the suture line has much in its favor. For this purpose, heavy processed silk, stainless steel or silver wire should be used.

The suture of facial wounds, or wounds normally visible is most satisfactorily performed with non-absorbable materials. The advantages of high tensile strength in small diameter and lack of reaction are nowhere more desirable. While horse hair is occasionally used for this purpose, but it has little superiority over fine processed silk and its lack of uniform strength is a disadvantage.

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RECURRENCE IN SEGMENTAL ENTERITIS FOLLOWING RADICAL RESECTION*

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IN 1909 Heinrich Braun[1] reported on "inflammatory tumors of the intestines whose recognition is important because of their similarity to new growths." Braun found that Moynihan[2] had seen the condition six times in three years and that Mayo Robson had seen it five times in twelve years, never, however, involving the small intestine. It was pointed out that these tumors bore no relation to tuberculosis or to true new growths.

Since the beginning of the century and before, surgeons have encountered this inflammatory type of benign intestinal tumor time and time again and were convinced in a vague sort of way that it was in a class by itself. Attempts at etiologic determination have been made, even up to the present, by isolated workers without success, although Felsen[4] has reported five cases associated with the cultural and serologic findings of bacillary dysentery. In general, however, very little differentiation was made in the heterogenous group of benign tumors of the intestinal tract, and they were all lumped together under the name nonspecific granulomata.

In October, 1932 the brilliant work of Crohn, Ginzburg and Oppenheimer[5] was published, establishing great order and much light in this confused, controversial field. These workers suggested the term "regional ileitis" for what they considered a new clinical entity strictly limited to the last portion of the ileum. An accurate description of the pathology was made, and several stages in the progress of the disease delineated. The most successful treatment was described as radical removal of the involved area, with ileotransverse colostomy and resection of the short-circuited portion of bowel. Subsequent reports, however, by Crohn and many other investigators[8,19,24] reveal definite proof that this same pathologic process manifests itself in various parts of the small and large intestine. The etiology is still undetermined.

As understanding of the pathology increased, various workers began to feel, and justly so, that the name given to the disease by the original authors was inaccurate. Many alternatives have been suggested in the literature. The disease was first called "terminal ileitis," but when manifestations were reported in the proximal ileum, Crohn, at Bargen's suggestion, changed the name to "regional ileitis." Shortly thereafter Harris, Bell and Brunn,[8] in reporting three cases, in one of which the jejunum was involved, offered the name "chronic cicatrizing enteritis." This was followed by the term "regional enteritis" by Brown,[11] and "segmental enteritis" by Lewisohn.[10] We believe the last to be the most descriptive term.

Segmental enteritis is a disease which suggests ulcerative colitis[10] in its course and manifestations, although Crohn and Rosenak[11] take great pains to show that there is a decided difference. The patient has a prolonged history of gastrointestinal upsets with remissions, which gradually lead to obstruction and surgical intervention. At operation in about 60 per cent of cases a mass is found in the right iliac fossa, the process most frequently, but not always, involving the last 20 to 30 cm. of ileum to the ileocecal valve. In advanced cases fistulae may be found connecting

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adjacent portions of bowel or occasionally extending to the anterior abdominal wall. Walled off abscesses may surround these fistulae and, if drained, may lead to intractable fecal fistulae.

The pathology has certain features which are fairly constant. Although many portions of the intestine may be involved, the inflammatory process most frequently affects the last part of the ileum, and in this part is more advanced distally than proximally. Microscopically, the picture is one of nonspecific inflammation. The mucous membrane may be destroyed and replaced by an atrophic layer of epithelium. Giant cells are occasionally seen and the submucosal layers are mainly involved. In the cases in which no appendectomy had been performed (50 per cent) the walls of the appendix have an involvment similar to that of the bowel but a normal mucosa.[5]

The disease appears to be fairly limited to young adults with a preponderance of males of the Jewish race affected. The period of illness extends over months or years with diarrhea, fever, loss of weight and anemia almost constant characteristics. The diarrhea is the chief complaint with two or four stools a day, usually containing mucus and often blood and pus. In the obstructive stenotic cases pain, visible peristalsis and vomiting are present. The characteristic physical findings listed are: (1) scar of a previous appendectomy; (2) a right lower quadrant mass; (3) evidence of fistulae; (4) evidence of intestinal obstruction; (5) emaciation and anemia. The signs and symptoms will correspond to the stage of the disease, which may early simulate acute appendicitis and later point to stenosis and fistula formation. The stage of stenosis and intestinal obstruction is the most common.

Although the diagnosis may be suspected clinically its ultimate establishment rests with the findings in the roentgenologic examination. The Roentgen findings, according to Jellen,[13] are as follows: (1) stenosis of the involved bowel segment, possibly with Kantor's string sign;[12] (2) non-visualization of the involved segment in some cases; (3) dilatation of the intestine proximal to the involved segment; (4) deformity of the cecum; (5) displacement of the normal small bowel by a mass in the right lower quadrant; (6) presence of fistulae; (7) evidence of associated colitis in some cases, with spasm, irritability, and localized hypermotility of the involved segment of bowel.

Segmental enteritis is to be differentiated from ulcerative colitis, ileocecal tuberculosis, fibroplastic appendicitis (or typhlitis), carcinoma of the large bowel, Hodgkin's disease, lymphosarcoma, actinomycosis, intestinal or mesenteric tuberculosis, sarcoma, and rarely, carcinoma of the small bowel.

The treatment of the condition is primarily surgical. Medical treatment is merely palliative and supportive, or preparatory for surgery. The surgical treatment of segmental enteritis has varied in the hands of different operators, and in the hands of the same operator, from simply opening and closing the abdomen to short-circuiting the involved bowel, with or without resection. From an examination of reports in the literature it appears quite generally agreed that radical resection, in one stage if possible, gives promise of resulting in the greatest number of cures.

Since accurate and sustained studies of the pathology and intestinal distribution of segmental enteritis, and its treatment by radical resection date back only eight years it is still too early to know definitely what prognosis radical resection actually gives. Koster et al.[14] first brought this question up in their excellent analysis in May, 1936, and at the end of the year Crohn[15] considered the same problem. In spite of Crohn's sanguine support of resection, citing thirty-two cases operated on by Berg with one death and no recurrences in the remainder, recurrences *are* taking place and reports of these are beginning to find their way into the literature. (Table 1.) The question arises, of course, as to whether

these are true recurrences or technical surgical failures.

We are concerned, in this paper, with the postoperative recurrences in segmental enteritis following radical resection. Although not all reported cases give sufficient details for analysis we have extracted from the literature all cases in point and summarize these herewith.

Probably the first reported recurrence following radical resection was described by Moschcowitz and Wilensky[16] in 1923, the description of pathology agreeing essentially with that outlined by Crohn and his associates in 1932. One year after a first resection it was necessary to resect another 12 inches of ileum proximal to the first anastomosis. In 1927 these same

TABLE I

REPORTED RECURRENCES FOLLOWING RADICAL RESECTION

No.	Author	Site of Occurrence	Area Resected	Interval	Site of Recurrence
1	Moschcowitz and Wilensky, 1923	Terminal ileum	Terminal ileum	1 yr.	12 inches terminal ileum proximal to anastomosis resected.
2	Coffen, 1925	Terminal ileum (?)	Terminal ileum (?)	5 mos. 8 mos.	(a) 24 inches terminal ileum resected. (b) Site? Third resection done.
3	Moschcowitz and Wilensky, 1927	Ascending colon	Lower ileum, cecum and ascending colon	5.5 yrs.	Proximal to previous anastomosis 12 inches ileum resected.
4	Crohn et al., 1932	Terminal ileum	Lower ileum, cecum and ascending colon	Not mentioned	Annular stenosis short distance proximal to anastomosis.
5	Homans and Haas, 1933	Terminal ileum	Lower ileum, cecum ascending colon	2 yrs.	Proximal loop of anastomosis.
6	Brown, Bargen, Weber, 1934	Terminal ileum and cecum	Terminal ileum and cecum	9 yrs.	Abscess and fistula. Site?
7	Musick, 1935	Terminal ileum	Terminal ileum	Not mentioned	Terminal ileum resection.
8	Shearer and Jackson, 1937	Terminal ileum	Terminal ileum and cecum	3 yrs. 8 mos. 9 yrs.	(a) Terminal ileum for 6 in. proximal to anastomosis. 10 inches ileum resected with part ascending colon and mesentery. (b) 12 inches of terminal ileum to anastomosis. Resection and ileotransversostomy.
9	Colp, 1938	Terminal ileum	40 cm. of ileum, cecum, and part of ascending colon	2 yrs.	Terminal ileum at anastomosis. 5 yrs. after first resection 18 inches more of terminal ileum removed.
10	Felger and Schenk, 1938	Terminal ileum and cecum	Terminal ileum, cecum, ascending and part of transverse colon	6 yrs. 3 mos.	Terminal ileum at anastomosis and a portion of ileum proximal with intervening "skip" area. 24 inches ileum and part transverse colon resected.
11	Felger and Schenk, 1938	Terminal ileum	Terminal ileum and cecum	17 mos.	8 inches terminal ileum.

workers[16] reported another case with the same pathology, but this time located entirely in the ascending colon. The cecum, ascending colon and terminal ileum were resected. About five and one-half years later the patient was operated on again and a mass was found proximal to the previous anastomosis. Twelve inches of ileum were removed and a new end-to-side anastomosis performed, using a Murphy button.

Coffen,[17] in 1925, reported three successive resections in one patient. This patient was a male of 20 years who, in 1916, was operated on for "appendicitis," appendectomy being done for "subacute inflammation." Abdominal exploration at this time was negative. About one month later the patient had an attack of abdominal pain, nausea and vomiting which subsided, but three weeks following this episode he was operated on for intestinal obstruction and 8 inches of "indurated bowel" were resected. Five months after this operation the patient was operated on a second time for an acute obstruction and 24 inches of ileum resected. A third resection was performed eight months following the second.

In the first report of Crohn[5] there is mention of an instance in which recurrent symptoms were accounted for by finding an annular stenosis a short distance proximal to the new anastomosis (ileotransverse colostomy). Apparently the resection was not sufficiently oral in location.

Homans and Haas,[18] in 1933, described a case in which recurrence took place after radical resection of lower ileum, cecum and ascending colon with end-to-side anastomosis of ileum to transverse colon. In this case the authors feel that lymph glands left at the mesoileum may have been responsible.

In 1934, Brown, Bargen and Weber[19] reported a case of radical resection which apparently was a "cure" until, nine years later, an abscess and fistula developed.

A year later, in 1935, Musick[20] mentions in general that "in several cases a distally resected ileum again became infected and it was necessary to do a second operation."

One of the best analyses and summaries of the entire segmental enteritis problem is contained in the report of Koster, Kasman and Sheinfeld[14] in 1936. In sixty-five collected cases of resection there was recurrence of the original lesion in ten cases, or 15 per cent. Of these, six patients were "cured" following a second operation, three cases were fatalities following a second operation, and one patient has had no further surgery to date (being apparently too close to recent surgery to be designated as a "cure"). In the past six years, since 1932, there have been about 200 cases of segmental enteritis reported. Radical resection tends to run about 74 per cent; sidetracking operations about 17 per cent, and in about 9 per cent of cases, mostly early, the abdomen was simply opened and closed.

In 1936, Connell[21] reported a rather interesting case history from his old records:

The patient was a female of 29 years.

First hospitalization, 1905. Chronic appendicitis had been present for two years and appendectomy was done at this time. The patient was symptom-free for one year, but then for the next six years she suffered from bouts of partial intestinal obstruction, occurring three to five times a year, with pain and swelling in the right iliac fossa.

Second hospitalization, 1912. The terminal ileum was thickened and enclosed in a fibrous sac. The membrane was removed, the ileum straightened out, and omentum placed over the bowel.

Third hospitalization, 1912, three months later. Ileosigmoidostomy was performed for chronic obstruction.

Fourth hospitalization, 1912, one month later. Enterostomy was done for acute obstruction.

Fifth hospitalization, June, 1913, eight months later. The lower ileum was markedly improved and localized jejunitis was the most striking finding.

Sixth hospitalization, November, 1913. Resection of the terminal ileum, ascending colon and one-half of the transverse colon was carried out. The gastroenterostomy and ileosigmoidostomy were functioning satisfactorily.

Up to 1925 the patient appears to have been all right with the exception of occasional gastrointestinal upsets.

Shearer and Jackson in 1937[22] reported two recurrences in a single patient.

First operation, March 31, 1923. Wide resection of ileum and cecum was done.

Second operation, November 15, 1926. The terminal ileum was involved in a process similar to the previous one. There was a well defined diseased area, beginning abruptly 6 inches above the line of the anastomosis with the colon and ending sharply at this last point. The terminal ileum was removed about 10 inches above, with the ascending colon and mesentery.

Third operation, September 12, 1935. The same gross pathology existed, well defined and sharply demarcated, beginning 12 inches above the last anastomosis and ending at the anastomosis. The mesentery was thickened and contained many enlarged lymph nodes. Another resection plus an anastomosis of ileum to transverse colon was performed. The patient was reported well fourteen months following operation.

In the first months of 1938 Colp[23] reported a case of recurrence:

1931, ileocecal resection, with removal of 40 cm. of ileum, cecum and part of ascending colon.

1933, *x*-ray showed irregularity of anastomosis of terminal ileum.

1936, patient began to complain of increasing abdominal pain.

1937, *x*-ray showed marked changes in the terminally anastomosed ileum, verified by operation. The ileum was divided again through healthy bowel and an ileosigmoidostomy performed. Eighteen inches of the previously anastomosed ileum was involved. The colonic portion was not involved.

CASE REPORTS

Case 1. Mrs. K. R., housewife, age 41, was admitted to the Cedars of Lebanon Hospital, on November 24, 1935, complaining of nausea and vomiting, diarrhea, abdominal pain and loss of weight. She had first noticed diarrhea and loss of weight in May, 1934. In November she began to have acute abdominal pain associated with diarrhea. She was hospitalized at the Brooklyn Hospital for two weeks—the suspected diagnosis being duodenal ulcer. The diarrhea returned and she was admitted to the Jamaica Hospital for study. *X*-ray showed no definite colonic pathology. There was diminished function of the gall bladder. Chest *x*-ray revealed evidence of primary parenchymatous calcified tuberculosis in the lower portion of the left upper lobe—apparently healed. An exploratory laporatomy done August 12, 1935 disclosed serous tubercles in the terminal ileum. Appendectomy was done. The postoperative diagnosis was tuberculous enteritis.

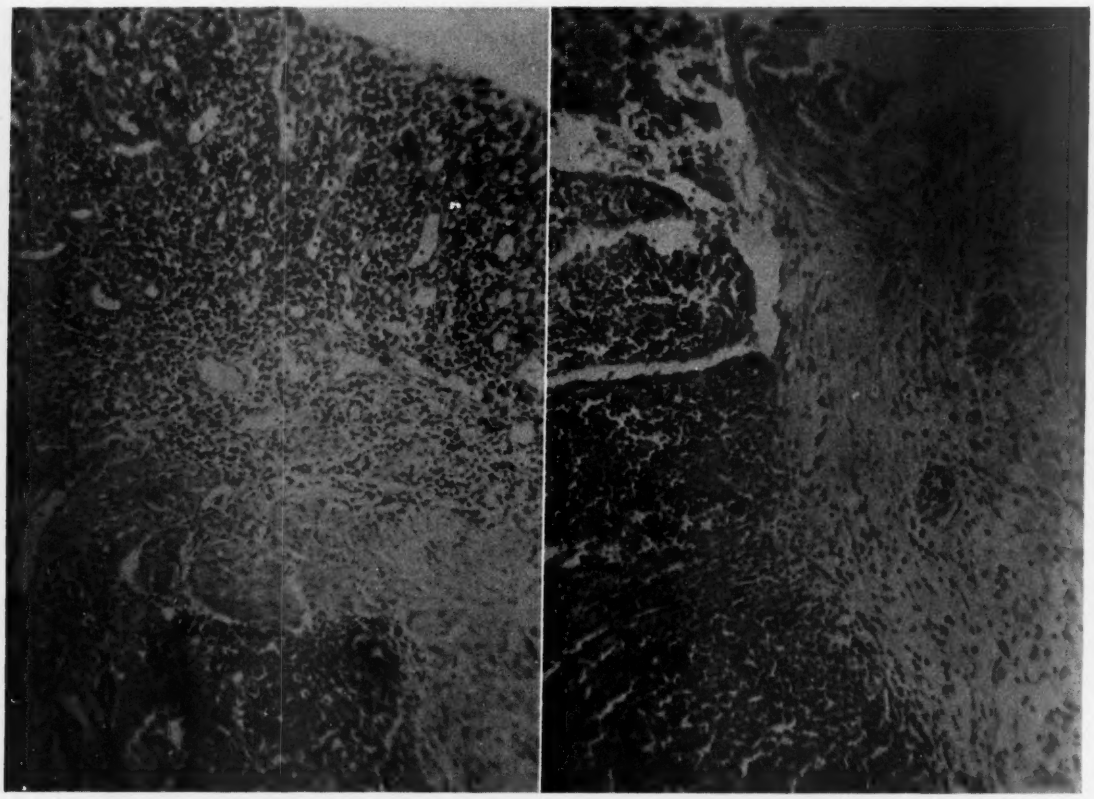
Following her discharge from the Jamaica Hospital, the patient was sent to a tuberculosis sanitarium at Duarte, California. The sanitarium questioned the diagnosis of tuberculous enteritis where there was no active pulmonary tuberculosis.

The patient was admitted to the Cedars of Lebanon Hospital November 24, 1935 for further study. *X*-ray showed the distal 4 inches of the ileum narrowed, the right kidney and colon ptosed, a lateral curvature of the spine, and phleboliths in the spleen. Barium enema showed ileocecal incompetency and narrowing of the distal 4 or 5 inches of the ileum. Sigmoidoscopic examination revealed no pathology up to 10 inches. The diagnosis was chronic terminal ileitis. Operation was done December 9, 1935. The terminal ileum was found to be hard, pipe-stem and the proximal ileum distended. Resection of the terminal ileum and cecum was done with ileocolostomy. The pathologic report was subacute regional ileitis. (Fig. 1.) The patient made an uncomplicated convalescence and was discharged to a convalescent home on January 1, 1936.

She was readmitted May 7, 1937 complaining of diarrhea, weakness, and loss of weight. She stated that she had continued to have diarrhea, had gained weight up to July, 1936 and then lost over 20 pounds. *X*-ray showed spasticity of the terminal loop of the ileum and transverse colon. Barium colon examination revealed patent anastomoses with narrowing of the terminal ileum of the pipe-stem variety. *X*-ray of the chest showed no active lesion. On sigmoidoscopic examination no pathology was found as far as the distal portion of the sigmoid. Operation on June 18, 1937 revealed the ileo colostomy functioning, the terminal ileum for 8 inches indurated and hard, with the presence of enlarged glands. A resection of the terminal ileum and the remaining portion of the ascend-

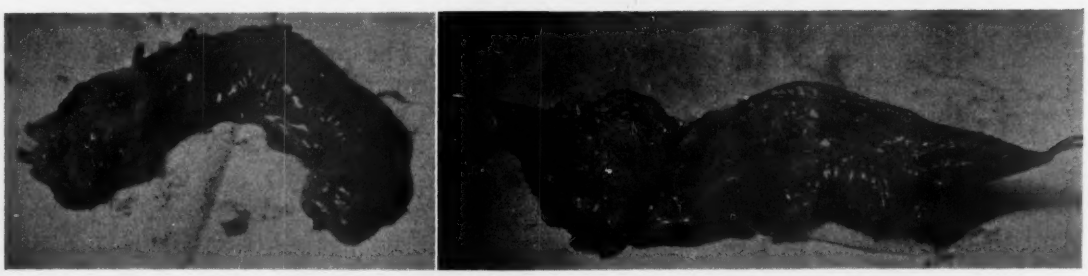
ing colon, together with a portion of the transverse colon, with side-to-side ileocolostomy was done.

of mucosa stuck up like small polypi above the areas of ulceration. Numerous lymph nodes were found at the mesenteric attachment



A B

FIG. 1. Case I. A, mucosa of ileum resected in 1935, showing erosion of epithelium and lymphoid hyperplasia. B, submucosa of recurrent involvement of ileum, removed in 1937, showing lymphocytic infiltration in granulation tissue.



A B

FIG. 2. Case II. A, recurrent larger segment measuring 26 cm., removed at operation May 27, 1938. B, specimen opened, disclosing thick edematous wall, narrowing and obstruction of lumen, normal wall and mucosa at each end of involved segment.

A pathologic report covering the 40 cm. segment of ileum showed that the middle 20 cm. were quite swollen, firm and almost completely obstructed. It was almost impossible to force fluid through the bowel. The wall of the gut was about 1 cm. in diameter and the lumen markedly narrowed. Through this area the mucous membrane was ulcerated and covered with a fibrinous exudate and surviving islands

opposite this segment of bowel. All of these were markedly enlarged, hyperplastic and, upon section, showed a wet, edematous surface. Grossly, the picture was one of ulcerative ileitis with characteristic thickening of the wall and section of the bowel.

Sections showed a segment of bowel wall which was very edematous and obviously infiltrated with round cells. (Fig. 2.) In places,

the mucosa was missing and was replaced with granulation tissue. In addition one saw numerous secondary abscesses in the submucosa.

The patient made an uncomplicated convalescence and was discharged July 9, 1937. Eighteen months after operation her condition

Fig. 3. Case 11. Recurrent smaller involved segment measuring 12 cm. Findings at operation May 27, 1938. Dilated, reddened indurated loop of ileum in the left hand of the operator with contrasting loop of normal ileum held on the right.

Fig. 4. Case 11. Specimen in Figure 3 opened, disclosing involved area with thickening of the walls, absence of mucosal folds and normal wall and mucosa beyond involved segment.

Two of the lymph nodes were sectioned and showed marked endothelial hyperplasia with dilatation of the sinuses and filling up of the sinuses with inflammatory cells.

Subacute ulcerative ileitis was the final diagnosis.

was excellent. She had no complaints, and was on a full unrestricted diet, with no abdominal symptoms. Her weight was 108 pounds, a gain of 25 pounds.

Case 11. E. S., female, age 18, was admitted to Cedars of Lebanon Hospital, on January 30,

1938, complaining of a tender abdominal mass and anorexia. Seven years before she had complained of weakness and loss of weight. She

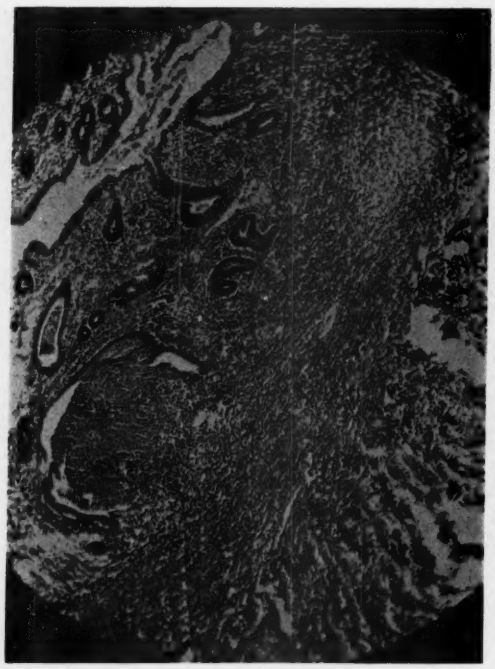


Fig. 5. Case II. Section from bowel, removed February 12, 1932, revealing marked infiltration of fibroblasts, lymphocytes, plasma cells, eosinophiles and leucocytes throughout the submucosa and mucosa.

was admitted to a hospital in Chicago in 1932 where an abdominal tumor had been discovered and operation done. A hard infiltrated mass involving the appendix, cecum and ascending colon was found. The clinical diagnosis was probable sarcoma. A resection of the terminal ileum, ascending colon and part of the transverse colon, with ileocolostomy was done. The pathologic diagnosis was "acute and chronic enteritis with involvment of mucosa, submucosa, muscularis and peritoneal surface, with production of a fibromyxomatous granulation tissue and marked proliferation of lymphadenoid tissue." (Fig. 5A.)

The patient was perfectly well until August, 1937 when she began to lose weight, had diarrhea, and later developed pain in the right lower quadrant. On January 15, 1938 she noticed an extremely tender mass in the right lower abdomen. She had frequent chills and fever, and upon admission on June 30, 1938, was extremely ill with a large fluctuant mass in the lower angle of the old right rectus incision. The abscess spontaneously ruptured several weeks after admission. Methylene blue given by mouth appeared in the dressings; the patient developed a fecal fistula. *X*-rays revealed recurrent ileitis. The fecal drainage gradually decreased and the temperature re-

Fig. 6. Case II. Section from recurrent segment removed May 27, 1938, showing marked infiltration of plasma cells and eosinophiles through the whole thickness of the edematous bowel.

turned to normal. The patient was sent to a convalescent home to recuperate and return for surgery.

After four months' convalescence the patient was readmitted on May 11, 1938. At operation May 27, 1938, a mass consisting of dilated reddened and indurated ileum about which the omentum was firmly wrapped was found. (Fig. 4.) Proximal to this involved portion of the ileum there was a segment of normal appearing ileum, but beyond there was another involved loop of ileum that grossly looked like ileitis. Resection of both involved areas of the ileum was done with a side-to-side ileocolostomy.

According to the pathologic report, the material consisted of two segments of small intestine, the larger measuring 26 cm. (Fig. 3.) Both were opened and the wall of the larger was thick and edematous. (Fig. 3B.) The mucous membrane contained numerous areas of ulceration. These ulcerations were elongated and looked like long clefts in the tissue. The small segment was 12 cm. long (Fig. 4) and the

central 8 cm. were involved in the same process of submucoal thickening, edema and small elongated ulcerations through the mucous membrane. The picture was grossly that of a chronic ulcerative ileitis.

Microscopic study (Fig. 5B) showed an intestinal wall which was markedly edematous. The mucous membrane in places was intact or missing, and there was marked infiltration of plasma cells, eosinophilic cells, and other round cells into the mucosa, submucosa and through the whole thickness of the edematous bowel. There was marked endothelial hyperplasia and in some of the large accumulations of lymphoid cells deep within the intestinal wall there were also germinal centers. In some places the mucosa was missing and was replaced by a slough of polymorphonuclear leucocytes. The edema, the filtration, especially of plasma cells and eosinophiles, and the endothelial hyperplasia fit in with the picture of regional ileitis.

Except for drainage and separations of the wound requiring secondary suture, the patient made an uncomplicated convalescence and was discharged August 28, 1938. Seven months later she appeared to be in excellent health and had no complaints. Bowel movements were regular, stools well formed and no diarrhea or abdominal symptoms were reported. She had gained weight, from 108 to 128½ pounds.

COMMENT

From a review of the cases reported it appears that following radical resection in segmental enteritis recurrences may be explained as due to one or more of the following reasons:

1. Resection insufficiently distant from the site of pathology. This category (as well as number 5) might include cases such as the one Crohn mentions in which surgery was performed through tissue which grossly appeared normal.

2. Permitting involved local glands to remain (Homans-Haas).

3. The existence of "skip" areas, pathologic areas separated by normal intestine (Harris, Ladd and others).

4. A special manifestation, or extension, of the disease ulcerative colitis.

5. Excision of an infectious, inflammatory lesion. This is not done any where else in the body, and is usually considered unsurgical.

6. Many of the recurrences have taken place after many years of apparent cure—up to ten years. Most of the reported cures have been followed for a relatively short time.

SUMMARY AND CONCLUSIONS

1. The clinical and pathologic aspects of segmental enteritis have been briefly reviewed.

2. Wide radical resection is the treatment of choice, care being taken to remove "skip" areas if present, and also involved neighboring glands.

3. Reported recurrences are cited and two cases of our own added.

4. Practically all recurrences in the reported cases are at the ileal site of the anastamosis.

5. Comment is made on the apparent reasons for recurrence following radical surgery.

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THE TREATMENT OF FRACTURED CLAVICLE IN THE PRESENCE OF KYPHOSIS BY A MODIFIED CLAVICULAR CROSS*

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THE elderly, stoop-shouldered man or woman, or the "hunch-back" presents a difficult problem in the use of the clavicular cross. The curving of the spine renders the cross ill-fitting and painful, and all too frequently it fails to give adequate fixation. Less efficient methods of fixation such as axillary pad, swathes, slings, tapes, and particularly recumbent treatment, are most certainly to be avoided in the very aged, in whom the rounded kyphosis is usually most severe.

These difficulties are overcome by the following simple modification of the clavicular cross, easily applicable to the kyphotic patient. It consists in principle of: (1) a posterior padded plaster shell; (2) the cross board incorporated in the shell; and (3) well-padded under-arm straps. It is extremely comfortable and efficient, and weighs only 4½ pounds.

METHOD

1. A trapezoid pad of thick saddle felt is cut to extend from the spinous process of the seventh cervical vertebra to the spinous process of the second lumbar vertebra. (Fig. 1A.) It is widest at the level of the spines of the scapula, where it extends within 1 inch of the acromion on either side. It tapers below to a width of about 4 or 5 inches.

This pad is carefully tailored to fit the patient, cutting out small wedges to adapt the pad exactly to the back. It may be sewed or held together with adhesive.

2. With the patient standing, the pad is temporarily fastened in place by 1 inch adhesive strips over the shoulders, suspender fashion. (Fig. 1A.)

3. A vertical splint of 8 inch plaster (eight to ten thicknesses) is applied to the felt and moulded to the back. (Fig. 1B.) Over it a transverse splint of the same thickness is applied and likewise moulded, forming a cross. (Fig. 1C.) As these splints set, two or three rolls of 6-inch plaster are applied to the entire felt pad, forming a padded shell of trapezoid shape. (Fig. 1D.)

4. With the patient still standing and the plaster shell in place, a thin board is cut for the cross, long enough to reach just inside the shoulders and wide enough to prevent axillary constriction, when the arm bands are in place.

5. With the board in the position it is to occupy, the plaster shell is marked along both top and bottom edges of the board with any sharp instrument. Remove the shell from the patient and proceed to fasten the board in place by first wrapping the board lightly in plaster, then building for it a buttress of plaster on the middle of the shell, accurately within the guide-lines. A few figure-of-eight turns secure it in place. Allow to dry about half an hour. (Fig. 1E.)

6. Meanwhile, one may make two soft, efficient shoulder straps as illustrated. A long absorbent abdominal pad of the type used in most hospitals is drawn inside a 3-inch stockinette approximately 2 feet long. This is faced with a strip of 2 inch tape of adhesive plaster and attached to the cross board, leaving one end free to be looped around the shoulder.

7. An abundant amount of sheet-wadding is placed over both shoulders running in figure-of-eight manner across the back

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and under each axilla. The fracture is reduced under local anesthesia, and while an assistant replaces the shell and holds it in position, the straps are looped under the axillae and fastened posteriorly to the cross board with safety pins and tape.

8. A strap of stockinette and pad is fastened across the front of the chest connecting the two loops and preventing their slipping off the shoulders.

9. A wide muslin bandage or elastic adhesive bandage about the lower thorax and the lower part of the cross completes the dressing. A vertical support to this band may be needed to prevent its slipping downward.

10. Position should be checked every other day for the first week, and should adjustments be necessary, they are simply made.

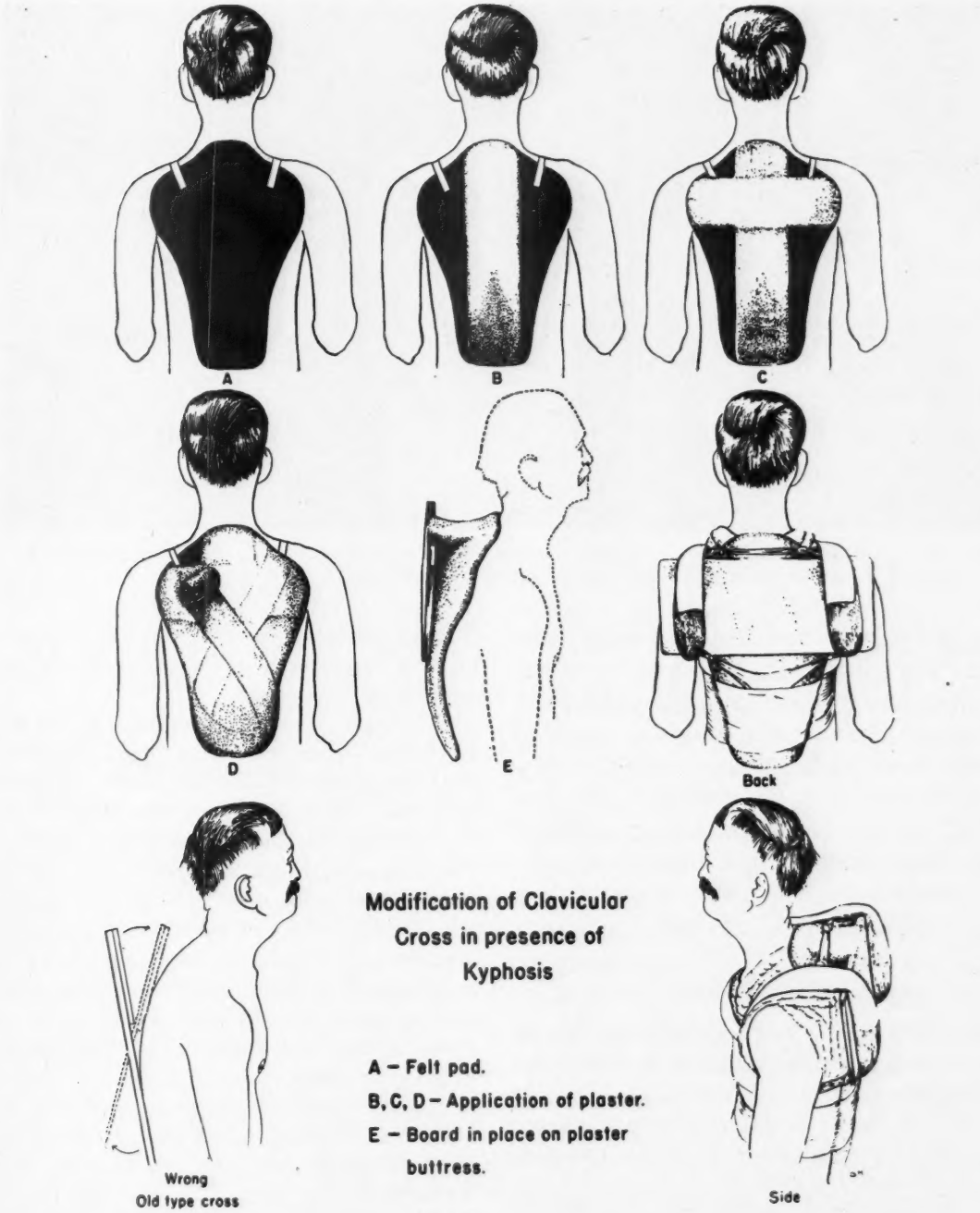

Fig. 1.

A review of the literature subsequent to the development of the author's method, reveals among all of the methods of treatment, only one article describing any similar procedure.[1] In this article is described a plaster slab applied in the form of a cross over a minimum amount of padding, and without the use of a buttressed cross-board so necessary to give upward, outward and backward position to the outer fragment. Furthermore, the prone position for the application of such a plaster slab seems ill-advised.

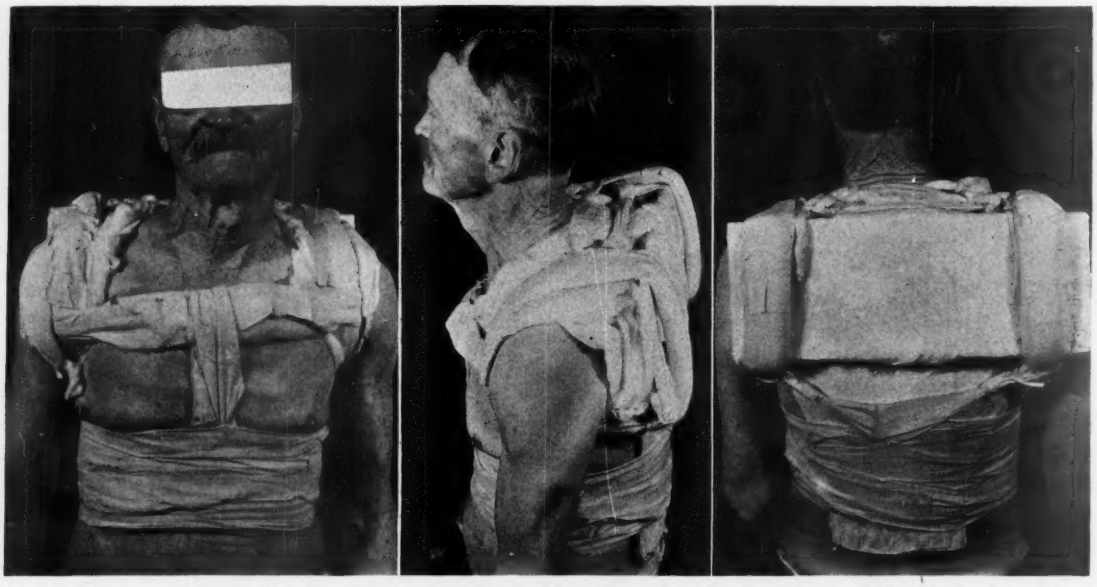

Fig. 2. Fracture of right clavicle. Modified cross had been in place three weeks, and the original position had been accurately maintained.

CASE HISTORY

Mr. J. B., aged 83, first entered the clinic on April 8, 1938, after having fallen to the street sixteen hours previous to entry, striking the point of his right shoulder. Physical examination showed a markedly kyphotic old man with an obvious fracture of the right clavicle. *X*-ray examination confirmed the diagnosis of fracture of the middle third of the right clavicle, with some comminution and marked displacement of fragments.

A conventional clavicular cross was applied and maintained for eight days. During this time the patient was rarely able to sleep because of the discomfort. The cross was difficult to maintain in its proper place, and the fragments were in poor position.

On April 16, the cross was removed and reddened pressure areas were noted over the vertebral borders of both scapulae. A modified clavicular cross was applied as described (Fig. 2), and the position checked every other day for the next week. The patient was very comfortable, able to be up and about, and the shoulders were maintained in good position. The cross was removed four weeks from date of injury, and a light figure-of-eight stockinette pad support was applied. The pressure areas noted above had healed. The fracture healed in good position.

Grateful acknowledgment is made to Dr. Nelson J. Howard, Chief of Fracture Clinic; to Miss Marjorie Marsten, artist; and to Miss Ruth Goss, secretary.

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AMMONIA AS A CELL PROLIFERANT AND ITS SPONTANEOUS PRODUCTION FROM UREA BY THE ENZYME UREASE*

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Ammonium Carbonate and Bicarbonate Stimulate Healing in Suppurating Wounds. In a recent report in this Journal the writer[37] has described the healing effects produced by ammonium carbonate and bicarbonate when used in treatment of chronic purulent wounds. These salts were found to be secreted by surgical maggots and to have the same therapeutic properties that the writer had previously reported for allantoin[32,34] and urea.[33,34]

When either of these ammonium compounds is applied to wounds with purulent drainage and with indolent, edematous tissues, a conspicuous change usually occurs in the condition of the wounds. The odor becomes less offensive, the thick discharge turns serosanguineous and the infection is reduced. Small islands of granulation tissue and blood vessels then begin to appear. The pallid areas become glistening and pinkish and this is usually followed in due course by a general growth of granulation tissue and final healing.

It is ammonia that brings about these results and a weak solution of ammonia (ammonium hydroxide) in water has the same effect. The carbonate part of the molecule is hydrolyzed into carbon dioxide and water.

The Results Appear To Arise through Stimulated Growth of Granulation Tissue. The cleansing and healing effects are not brought about through any solvent or direct bactericidal action, because in the concentrations used, namely from ½ to 2 per cent, these ammonium salts do not possess such properties. The pyogenic infection tends to disappear, it is true, but at the same time a vigorous growth of granulation tissue with blood supply usually forms in the wound—a condition unfavorable for bacterial activity. Effects are obtained with these ammonium compounds which are not accounted for on the basis of direct reduction of the infection, but which apparently can be explained through the proliferation of healthy granulations in the wound.

Billroth[8] in 1865 showed that such tissue protects the wound against the entrance of septic substances and infections. Afanassieff[1] later noted that it also secretes a bactericide. Arey[4] stated that granulation tissue "presents the ideal conditions for a defense against infections; this includes marked hyperemia, active exudation and local leucocytosis . . . "

In wounds with extensive indolent tissue, it is not uncommon to find small areas of new tissue a few days after the ammonia solution is applied. Such growth implies numerous and successive cell divisions in that period. Other factors besides ammonia could, of course, play a part in this proliferation. The mere technique of applying the treatment favors spontaneous healing. This includes the daily application of a wet pack to the wound, which is the way the ammonia solution is applied; frequent irrigations that mechanically remove debris and cleanse the wound; rest in bed. If these alone were successful in stimulating healing in chronic purulent wounds, ammonia could be considered inert. A common observation is that without changing any part of the treatment except adding

* Contribution from the Bureau of Entomology and Plant Quarantine, United States Department of Agriculture.

ammonia, such wounds frequently respond with healing.

Ammonia Is Utilized by Animal Tissues in Their Metabolism. A recent discovery of considerable value in this discussion is that animal tissues are capable of using ammonia in synthesizing amino acids which are essential in tissue growth. Euler et al.[12] were among the first to make such a report. They showed that bullock and hog liver preparations can synthesize glutamic acid from the α-keto acid and ammonia. Rittenberg et al.[31] have gone still further and have shown that rats can synthesize not only glutamic acid but also arginine, glycine and aspartic acid. They fed the ammonia to rats in the food and later found that it had been taken up and incorporated into various tissues. These investigators were able to trace the identical ammonia that they had fed, as they had prepared it with isotopic nitrogen.

Glutamic acid, mentioned above, is not only essential itself in the growth of new tissues but, according to Euler,[12] it also occupies a central position in the general amination of keto acids to amino acids.

Previous to these studies by Euler, Rittenberg and their associates, the synthesis of amino acids was believed to be a function of plants only. Their work has therefore shown the existence of another fundamental similarity between the plant and the animal cell. Ammonia and certain ammonium compounds have been known for a long time to have nutrient and growth stimulating effects in plants.

Ammonia from Protein Metabolism Is Not Necessarily Utilized Directly. Ammonia formed during the metabolism of proteins might be assumed to be directly available to the cells according to their needs. Certain difficulties appear, however, in the way of this simple and easy method of obtaining it. First, ammonia from that source could be inaccessible. Its occurrence in any given tissue does not imply that it is present in or available to every cell when needed. Secondly, while each cell produces some ammonia during its own metabolism, no proof exists of course that its protoplasm is ready at the same time to reutilize it. The protoplasm may be regarded as rejective and receptive of ammonia, as of other materials, according to its requirements. Moreover, the experiments of Rittenberg et al.,[31] already referred to, do not demonstrate that the rats utilized the ammonia directly from the food.

Ammonia Is Producible by the Intracellular Enzyme Urease. There is another source of ammonia, through the action of the enzyme urease upon the common metabolic product urea. Little consideration has been given to ammonia from this source; but all the essentials for its formation have been recorded for both plant and animal tissues. Also since this enzyme is readily activated and inhibited, there is the possibility of the control of its activity by the individual cells and the production of ammonia as needed. Three interrelated units are, therefore, concerned in this discussion, namely, the intracellular enzyme urease, the inert nitrogenous product urea, and ammonia which is reactive and rich in available nitrogen. Together they comprise a system in living tissues and it is with their potentialities that this investigation deals.

Urease Is Elaborated during Cellular Multiplication. Brunel and Échevin[9] in France were apparently the first to note the activity of urease at the time of cell proliferation. In 1937 they determined its activity in dormant and in germinating seeds of the plant *Agrostemma githago*, and they stated that this enzyme is absent from the dry seeds but that it is elaborated in considerable quantities during the early growth or sprouting process.

Granick[17] found that the concentration of urease in the jackbean is highest when the cells of the young structures are rapidly dividing and that it decreases markedly as the tissues grow older. He discussed both the formation and the destruction of this enzyme by the cells.

Coincident with rapid cell proliferation in the fungus *Aspergillus niger*, the activity of urease increases greatly, according to Bach.[7] When growth of the tissues is completed, the production of urease diminishes rapidly. In confirming this work, Brunel[10] noted that the concentration of urease had fallen over 80 per cent at maturity of the plant.

Marked variations in the production of urease were determined by Robinson and Wilson[36] during pupation of the surgical maggot *Phormia regina*. Extraordinary tissue changes occur in this pupal period, as the headless maggot develops into the winged fly. The larval organs are autolyzed and an entirely different set of adult organs is formed. Early in this period the concentration of urease increases 57 per cent and is followed shortly by a still greater decline. The values then rise again and finally fall abruptly when growth is completed.

It is noteworthy that this is a non-feeding period and that the tissue changes are made at the expense of existing material. Owing to the great economy necessary during that time the marked activity of urease probably renders the nitrogen in the urea available in the form of ammonia.

Occurrence of Urease in Living Tissues. The discovery of urease in living processes was first made in bacteria, and many species are now known to produce this enzyme. Then followed a search for it among plants with the result that it has been found in every division of the plant kingdom.[16] Among animals, urease is also widely distributed from insects to human beings and in various organs. This search, unfortunately, has not been intensive as little value has been attributed to its presence in the organism.

Albrecht[2] in 1921 detected urease in the digestive tract and in the muscles of several kinds of marine mollusks. Luck[24] discovered it in the gastric mucosa of the dog, cat, bullock, sheep and goat. He stated that although this enzyme is contained in the animal body, little can be said about its physiologic importance.

Loeb and Bodansky,[22,23] in a study of the king crab Limulus, found urease in considerable amount in the amebocyte tissue, blood, muscles, and eggs, and stated that the blood can easily extract it from the amebocyte tissue. They concluded that no statement could be made at that time on the function of this enzyme. Przylecki[30] found urease in snails, mussels, earthworms and crabs.

Steppuhn and Utkin[38] noted the presence of urease in the livers of rabbits, horses and dogs. They suggested that it entered the body with the food, and felt that in any case the enzyme appears to have no significance. Robinson and Baker[35] showed that it can be elaborated by animal tissues: they determined its presence in sterile blowfly maggots which had been reared upon autoclaved, enzyme-free food.

Majorow[25] found urease in hypophysis and adrenal glands, and remarked that the ovaries are particularly rich in this enzyme. Cardin[11] noted its appearance in the gastric mucosa of the human fetus about the fourth month. He believed its activity is limited to the stomach in connection with the neutralization of excess HCl.

In 1937 Tauber[41] referred to evidence of the occurrence of urease in animal tissues but believed that it had not been shown to be of importance. It is since that time, of course, that much of the information used in the present investigation has become available.

The Enzyme Urease Is Specific for Urea. A fact that seems to be significant is that urease can attack only urea. It is so highly specific for this metabolic product that it cannot react upon even closely related compounds. This peculiar specificity of urease early noted by Takeuchi[40] and Armstrong et al.[5] has since been well confirmed by others. It therefore appears worthy of consideration that urease, a complex protein, with a molecular weight of 483,000 according to Sumner,[39] exists in all types of living organisms and with no demonstrated use but to hydrolyze urea.

Metabolic Urea Is Present in All Animal Tissues. There is no lack of urea in animal tissues for urease to react upon; it appears to be present in all tissues and liquids. According to Andresen,[3] Marshal and Davis,[26] and Fearon,[13] urea occurs in approximately uniform concentration throughout the body. The content in the blood of man is from .02 to .03 per cent. Fluctuations in the urea content of the lymph and blood cause corresponding changes in the tissues. Urea is very soluble and diffusible, and the cells can absorb it readily; Gryns[19] finds that solutions of urea readily permeate the membranes of all kinds of cells; and Peters and Van Slyke[27] state that urea can pass through membranes without apparent resistance.

Urea as a Reserve Supply of Ammonia. Urea is known to be an inert substance physiologically and a means of removing excess nitrogen from the body in the urine. The peculiar properties of urea fit it also for another function, namely, a source of ammonia. Physiologic processes are remarkably efficient, and it is therefore difficult to comprehend that urea which so easily penetrates to all the tissues should be excretory only. A product low in solubility and diffusibility would be more effectively excreted without first premeating the cells.

"The high nitrogen content (46.6 per cent), the neutral and nontoxic characteristic, and the ready diffusibility of the small urea molecule" observe Peters and Van Slyke,[27] "all seem to render it peculiarly adapted to serve as a vehicle for the excretion of waste nitrogen" and, it might be added, as a supply of ammonia to the cells.

Ammonia constitutes a large part of the urea molecule and is constantly being formed during digestion and metabolism. Normally, 0.3 mg. of ammonia is said to be contained in 100 c.c. of blood. In excess it is injurious to the tissues and therefore it does not accumulate but is built up by the liver into the stable and harmless urea. This process removes ammonia as such from circulation. If urea should have only one function, namely the removal from the body of a waste product of metabolism, it would be a strange situation to have all the tissues of the body bathed perpetually in an excretory substance.

It is not a new conception that the organism has some means of storing ammonia in a harmless form. Prianischnikow[28,29] in 1924 regarded asparagin as serving this purpose in plants. He went one step further and drew an analogy between asparagin in plants and urea in animals. He believed, however, that there is a difference between them in that the urea is excreted by the animal as a waste product whereas the asparagin is available to the plant.

The Concentration of Urea in the Tissues Is Suitable for Urease Activity. The weak solution of urea in mammalian tissues is favorable to high activity of urease. Armstrong and Armstrong[6] discovered in 1913 that urease works best in weak solutions, and Van Slyke and Cullen[42] found later that it reacts at its maximum efficiency in concentrations as low as 0.08 per cent. This applies to pure solutions of urea, and the presence of certain physiologic products slightly increases the activity of urease so that it reacts well in the normal urea content of the tissues. For instance, Jacoby[20] found that blood serum accelerates the activity of urease.

The Hydrolyzing Property of Urease Is Great. The potential capacity of urease to break down urea appears to be enormous. Van Slyke and Cullen[42] showed that one molecule of the enzyme can disrupt molecule after molecule of urea continuously without any measurable loss of time between contacts. Granick[18] has recently calculated that one urease molecule is able to split 2,000 urea molecules in one second. Fortunately, this rapid rate of hydrolysis of urea does not occur normally in living tissues; it requires not only a continuous supply of urea but also an active state of the enzyme and a rapid removal of the breakdown products.

Inhibition and Activation of Urease. Both urea and urease having been found in animal tissues, it follows that a coexistence of the two would imply a rigid control of urease activity. Numerous experiments in vitro have shown that urease is readily accelerated and retarded, but evidently not a great deal is known about its spontaneous control.

Armstrong et al.[5] found that the two end products of the enzymic breakdown of urea, namely ammonia and carbon dioxide, strongly affect the activity of urease. The authors say: "While the activity of urease is much reduced by ammonia, it is increased in a remarkable manner by carbon dioxide; in other words, the two products affect the activity of the enzyme in opposite ways—a result altogether without precedent." This has since been amply confirmed.

It is especially noteworthy that ammonia thus appears to have a dual rôle. On the one hand, it is used by the protoplasm in the building up of amino acids essential in cell proliferation, and this is supported by the ability of ammonia to stimulate growth of granulation tissue in wounds. On the other hand, ammonia is an inhibitor of urease activity and can react, conceivably, to limit its own supply within the cell. This would constitute an automatic control of ammonia formation from urea; for normal cells obviously require less ammonia in their resting period than when proliferating. It would be interesting to know if the more rapid rate of cell division in malignant growth is associated with sustained ammonia receptivity and urease activity.

A Urease-Urea-Ammonia System in Living Tissues. From the experiments of numerous investigators, Kiesel[21] in 1927 concluded: "Urea without being broken down is useless for plants, whereas in the presence of the enzyme urease it possesses an excellent nutritive value. Urea can be assimilated only after hydrolysis by living cells . . . The importance of urease becomes clear in cases where the nitrogen of urea re-enters the protoplasm . . . One might not go wrong in expressing the same viewpoint for the animal kingdom."

Fosse, who discovered in 1912 that plants produce urea,[14] has also commented upon the relationship between urea and urease, as follows:[15] "The rôle and utility of urease in plants, unknown before our researches because the formation of urea in their cells was not known, consists precisely in the destruction of urea which is non-assimilable and the production of two excellent foods: carbon dioxide and ammonia. That is why, as we have shown, the same plant contains simultaneously urea and urease, and is the seat of two inverse phenomena, the formation and the destruction of urea."

The available data bear on the occurrence of this phenomenon in plants. The possibility of its occurrence also in animal tissues has evidently not been given as much attention.

Negative results on the presence of urease in certain animal tissues, reported by a few authorities, require investigation. For instance, Przylecki[30] found urease in earthworms but none in the related leeches. Owing to the minute amount of urea in any cell and the potentiality of one molecule of urease to hydrolyze many thousands of molecules of urea, it is probable that the enzyme is present in the cell in a microscopic amount in some cases, and could escape detection except by microanalysis. Also, as noted by Wilson,[36] the period of incubation with urea should be sufficient to allow the cells to autolyze and thus release the enzyme which of course is intracellular.

SUMMARY

A recent report by the writer that ammonium carbonate and bicarbonate stimulate growth of granulation tissue in wounds is correlated with other reports that ammonia is synthesized by animal tissues into several amino acids essential in cell proliferation.

In normal utilization of ammonia in growth processes, ammonia produced dur-

ing digestion and protein metabolism is not necessarily accessible to the cells. Ammonia-reception and ammonia-rejection by the cells are discussed.

Ammonia arising from the action of the enzyme urease upon metabolic urea appears to be available to the tissues. All the essentials for its formation have been recorded for plant and animal tissues. The ease with which the enzyme can be activated and inhibited in vitro raises the possibility of its spontaneous control.

Numerous reports of the occurrence of urease in living tissues are cited, and investigations by the writer and others on the activity of urease at the time of cellular multiplication are discussed.

The peculiar specificity of urease for urea, the presence of urea in all tissues and cells, and the breakdown product ammonia are correlated.

The general conception that the function of urea is to remove ammonia from the body is contrasted with the possibility that urea is also a reservoir or source of ammonia. A postulation of this type for plants is cited.

The question of automatic control of urease activity, through the inhibiting effect of its own end product ammonia and the reutilization of ammonia by the protoplasm, is mentioned as a possibility.

The conception of the reaction of urease upon area in living tissues thus producing the nitrogenous product ammonia essential in tissue growth, is not new. Such a system has been described twice.

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THE VALVES AND ANASTOMOSES OF THE HEMORRHOIDAL AND RELATED VEINS*

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HEMORRHOIDS are the most common of all rectal disorders and, in association with their complications and sequelae, are of considerable clinical importance. They are essentially varices of the hemorrhoidal plexus. The study of the direction and rate of blood flow in the hemorrhoidal plexus is as essential in hemorrhoids as is the study of the veins of the lower extremity in varicose veins. The presence and location of valves and the degree of anastomosis occurring in the normal individual is the foundation for the study of the abnormgl rate and direction of venous return.

The venous plexus in and around the rectum drains into both the portal system and the inferior caval system of veins. The submucosa of the rectum from the level of the anorectal line to the rectosigmoid junction, as well as the rectal wall above the peritoneal reflection, is drained by the superior hemorrhoidal vein, a tributary of the inferior mesenteric vein and a component of the portal system. The rectal wall below the peritoneal reflection is drained by the middle hemorrhoidal veins, and the anal canal and the area immediately surrounding it is drained by the inferior hemorrhoidal veins. Both the middle and the inferior hemorrhoidal veins are tributaries of the hypogastric veins and are therefore components of the inferior caval system.

While anatomists have agreed upon many of the anatomic facts in the study of this region, the proctologists have shown considerable disagreement as to the degree of anastomoses and as to the presence of valves in the hemorrhoidal veins. For the foregoing reasons, it was felt that a study of the veins of the rectum and accompanying collecting veins would be profitable.

REVIEW OF LITERATURE

In 1877 Duret[9] studied the rectal veins by injection methods and described small collecting veins in the anal canal, located both under the anal skin and in the substance of the anal sphincter. He also described the connections between the superior hemorrhoidal vein and those of the plexus of the prostate, seminal vesicles and bladder.

Zuckerkandl,[33] in 1878, described perforating veins at the anus and suggested that, since they could be injected from their central ends, they did not possess valves. He also described the connections with the prostatic plexus and the middle sacral vein.

Gay,[12] in 1882, during an autopsy of a patient with hemorrhoids, injected the veins and found that the prostatic plexus could be filled from the internal iliac vein, but that this injection did not reach to the superior hemorrhoidal veins. Retrograde injection of the superior hemorrhoidal vein filled its tributaries to the anorectal line, but this injection "did not even approximate the prostatic plexus." As a result of these injections, he decided that there was no connection between the portal and caval systems around the rectum.

Quenu,[23] in 1892, stated that, since the injection of the submucosal network from the caval system was very difficult, there must be valves in the inferior hemorrhoidal veins so placed that they would hinder passage of blood from the caval to the

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portal system, but would favor passage in the opposite direction. He also demonstrated the connection between the two systems in the peri-sphincter tissue as well as by smaller veins in the anal canal. His injections of the superior hemorrhoidal vein showed the injection mass in the prostate and the seminal vesicles.

In 1908 Ball[3] said: "I have not, however, by injection been able to demonstrate the free anastomosis between the two systems, which has been described by some authors."

Cripps[6] found that the hemorrhoidal plexus could be injected from the inferior mesenteric vein, but that this injection did not pass into the iliac veins. He believed the connection between the two systems very slight and that if it did exist it allowed flow only toward the iliac veins.

Allingham[2] quoted the brief report of Verneuil and stated that there was little or no connection between the several hemorrhoidal veins.

Tuttle[29] believed that the connection between the two systems was so narrow as to be almost imperceptible in early life, but that in old people who had been constipated and had hemorrhoids, injection of the superior hemorrhoidal vein filled the inferior hemorrhoidal plexus.

Goodsall[13] and Miles thought the connection between the middle and the other hemorrhoidal veins very slight.

Cooke,[5] Lockhart-Mummery,[19] Yeomans,[32] Pennington,[20] and Gant[11] say that there is a free communication between the two systems in the region of the rectum. The anatomists Waterson,[30] Robinson,[24] Piersol,[21] Lewis,[18] Tandler,[27] Senior,[25] and Testut[28] all describe a free communication between the two systems at the rectum.

In considering the question of the presence of valves in the hemorrhoidal veins, Senior[25] notes that the superior hemorrhoidal vein, being a tributary of the portal system, is without valves. Piersol concurs that the portal veins are without valves, "although the opposite view is held by some."

Crisp,[7] in 1855, reported finding valves in the portal systems of many lower animals and Franklin,[10] in 1926, found valves in the portal vein of the sheep.

Hochstetter,[15] in 1877, saw valves in the portal systems of lower animals, in newborn infants and less frequently in adults. He studied the veins of the gastric region and reported that the valves were constructed in the same way as those of the caval system, but were so slight that they were easily forced by injection. He concluded that these valves atrophied with advancing age.

Bryant,[4] in 1888, confirmed the findings of Hochstetter and reported finding valves in the veins of the human intestine. Wilkie[31] demonstrated the presence of valves in the tributaries of the gastric, omental and colic veins of animals, infants, children and adults and suggested that, since they were less numerous in the aged than in the newborn, they were undergoing a retrograde metamorphosis. Neither of these authors reported specifically on the tributaries of the superior hemorrhoidal veins. Lewis, Robinson, Porier,[22] and Testut all state that there are valves in the portal system in the embryo and newborn, but that they atrophy in the adult.

Valves were stated to be present in the inferior hemorrhoidal veins by Agnew[1] and Cook. Senior, in discussing the tributaries of the hypogastric vein, states that the superior and inferior gluteal veins have valves as do the lateral sacral and iliolumbar. He does not mention the middle or inferior hemorrhoidal veins.

OBSERVATIONS

Material. Among the comparative animals, the material consisted of three dogs, examined in the recent state, and one macaque rhesus. In the human the veins were examined in the cadavers of eighteen infants and eight adults. There were also examined four specimens removed at autopsy. The adult human bodies were intact or partially dissected prior to the

examination. Both male and female human specimens were used.

Methods of Study. The veins of these specimens were examined by direct dissection and by microscopic examination of sections. Injections of Roentgen opaque and colored masses, using gravity or thumb pressure on the ordinary Luer glass syringe, allowed study of the vessels by stereoroentgenogram as well as by making transparencies after the method of Spalteholz.[26]

FIG. 1. Valve at the junction of the superior hemorrhoidal and last sigmoid veins in the newborn. (× 330.)

Observations on Animals. In the dog the portal vein tributaries are well provided with valves. None were found in the main portal trunk, but the ostia of the splenic, gastric and pancreatic veins were all guarded. The vena breva of the stomach contained valves as well as the small veins of the intestines. There were many atrophic or incompetent valves present in the various vessels. At the junction of the superior and inferior mesenteric veins there were arcuate white lines visible on the vein walls but no leaflets were present. The common iliac veins contained valves in some instances, but the external iliac veins were always protected, as were the hypogastric trunks and all the mouths of the tributary veins emptying into them. Injection of the portal system demonstrated valves competent to resist back pressure in the gastric and colic small veins. Of special interest was the finding of a competent bicuspid valve in the distal portion of the superior hemorrhoidal vein in two of the three dogs examined. This valve was capable of resisting pressure great enough to allow injection of the entire bowel mucosa above the site of the valve.

FIG. 2. Retrograde injection of the inferior vena cava of the newborn showing competent valves in the veins of the pelvis that prevent filling of the anorectal region as well as the vesicoprostatic plexus. Great pressure causes filling of the sacral and entire vertebral plexus and later the inferior cava and renal vein above the site of injection.

In the monkey there were no valves present in the larger tributaries of the portal system, and as the animal had died of peritonitis the bowel wall was so involved that study of the smaller tributaries was impossible. It was possible to inject the superior hemorrhoidal vein and the colored mass was easily visible down to the ano-

rectal junction but did not extend across the anorectal line. With the left common iliac clamped the left femoral vein was injected with a considerable amount of force and two small inferior hemorrhoidal veins were filled so as to become visible on the perineum on the left side. Dissection of the pelvis then showed that there was some discoloration of the rectal wall on the left side from the injection of the femoral vein and that this extended into the wall of the rectum and into the submucosa above the anorectal line in several places. Apparently there was some connection with the middle hemorrhoidal group of veins that allowed this to happen. The opposite side of the pelvis showed no filled veins and demonstrated the absence of a free anastomosis between the veins of the two sides of the pelvis. At a later time it was observed that in an experiment in which Dr. Batson was injecting the deep dorsal vein of the penis of a living monkey with thorotrast, the internal iliac as well as the external iliac vein could be filled but that the veins from the prostate and outside the rectum—the middle and inferior hemorrhoidal veins—did not fill as did the lateral sacral and obturator veins. This suggested that the mouths of these tributaries were guarded by valves.

Fig. 3. Injection of the portal system in the newborn. Great pressure was used and the superior hemorrhoidal vein filled but the middle and inferior hemorrhoidal veins did not.

Observations in the Newborn. The portal vein and its larger tributaries contained no valves. In several bodies there were white lines at the junction of some of the larger tributaries, but no cusps were present. It was noted in four bodies that lines were located at the junctions of the superior hemorrhoidal with the last sigmoid vein. In one specimen there seemed to be a competent valve here that would resist retrograde stroking and this was ligated and removed. Microsections made by the Department of Pathology demonstrated the competent valve at this point. (Fig. 1.)

In general the valves of the newborn were more constant in their locations in the pelvic veins of the inferior caval system and

it was also noted that these were always more competent to resist retrograde injection that was the case in many of the

Fig. 4. Injection of the deep dorsal vein of the penis of the adult with filling of the vesicoprostatic plexus and the large veins on the lateral walls of the pelvis. There is no filling of the veins in the anorectal region.

corresponding veins in the adult. Figure 2 shows an *x*-ray film made after the injection of the inferior vena cava. There are valves in the common iliac veins that resist pressure sufficient to fill the entire vertebral plexus. In no instance, using both fine and coarse masses, was it possible to fill the inferior or middle hemorrhoidal veins in a retrograde direction by injection of the femorals, the vena cava or the internal iliac veins. No color would appear in the prostate or rectum although the sacral veins could be filled by exerting considerable pressure.

Injection of the portal vein of the newborn would always first fill the gastric region, the spleen and the pancreas. In some cases there were bulges at the openings of the tributary veins along the greater curvature of the stomach and along those of the colon. Slight increase in pressure would always force the mass past these areas. The entire system could be filled, but by the time that the mass would appear in the lower colon and rectum the entire bowel wall of the upper intestinal tract would be injected. (Fig. 3.)

Anastomoses between the portal and caval systems around the rectum were demonstrated in only a single instance. The prostatic plexus and the sacral veins were filled only after great pressure was used and then the veins filled so poorly that they did not show on *x*-ray examination and were only found by dissection.

In one instance, where filling of the pelvic veins occurred following injection of the portal system, a dilute india ink mixture was used. The filling was not observed as the fluid was flowing by the gravity method under a pressure of 150 mm. of water. It was only after the injection had been completed that the outer rectal plexus was seen filled and the possibility could not be eliminated that it might have filled through some other upper abdominal route since the vertebral plexus was also filled.

The valves at the mouths of the tributaries of the hypogastric vein were not shown by *x*-ray examination as the veins were so small that they could not be satisfactorily demonstrated. However, it was possible to show that those veins did not fill after injection as they should have, had there been an avenue for retrograde flow unimpeded by valves.

Observations in Adults. One was struck by the many variations in size and location of valves in the pelvic veins in the adult. In most instances when the right and left halves of the pelvis were examined, there was decided difference in the two sides of the same body. In general there were no valves in the inferior vena cava, the common iliac and the hypogastric veins. There was a valve present in the external iliac vein in many cases, but in no instance was it competent to resist the back flow of blood caused by stroking and it never

blocked the injection mass. There was usually a valve below the femoral junction and at the junction of the obturator and would drain into larger veins on the lateral walls of the pelvis; the external iliac vein only rarely, the internal iliac vein almost

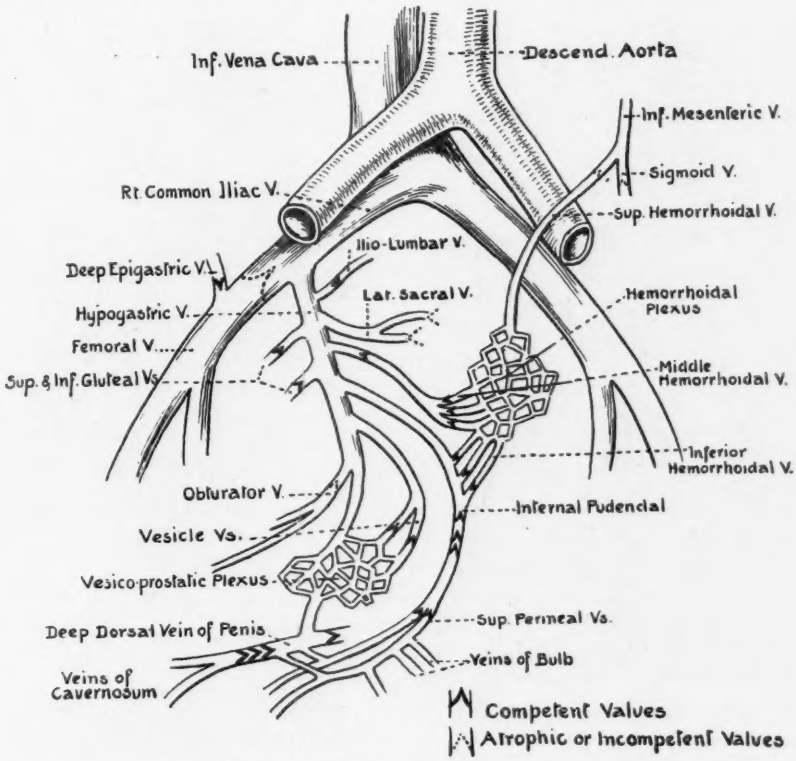

Fig. 5. Diagram of the pelvic veins showing the usual sites of competent and incompetent valves, as demonstrated by dissection and injection.

the deep epigastric veins. The tributaries of the hypogastric vein were protected by competent valves in almost all instances.

Injection by way of the deep dorsal vein of the penis which usually contains several sets of valves caused no filling of the middle or inferior hemorrhoidal veins. The valves at their mouths could be seen as definite bulges on *x*-ray examination. Valves were also noted guarding the superior and inferior gluteal, the iliolumbar, and the vesicle veins. The internal pudendal vein was sometimes guarded by competent valves and sometimes not.

The gross dissections demonstrated valves in the pudendal vein regularly, but they were apparently not always competent. In no instance was it possible to fill the hemorrhoidal plexus or prostatic plexus by injection of the femoral vein. The prostatic plexus could be filled by injection of the dorsal vein of the penis and this constantly, and the lateral sacral vein in varying degree almost constantly. The rectum was never injected, not because there was no connection with the hemorrhoidal plexus from the middle or inferior hemorrhoidal veins, but because the distal ends were protected by valves that did not allow them to fill. (Fig. 4.)

Dissections showed that there were frequently several sets of valves in the internal pudendal and at the mouths of the superficial perineal veins as well, even though these could not be injected because of the valves more centrally placed. It might be well to add that there were also valves at the smaller tributaries of the gluteal veins as well as in the subcutaneous veins of the scrotum and perineum. These were well marked and apparently competent. This finding shows that not only are the mouths of the main tributaries of the hypogastric vein protected but also the smaller tribu-

taries of these veins. The diagram in Figure 5 shows the locations of the valves found in the pelvic veins.

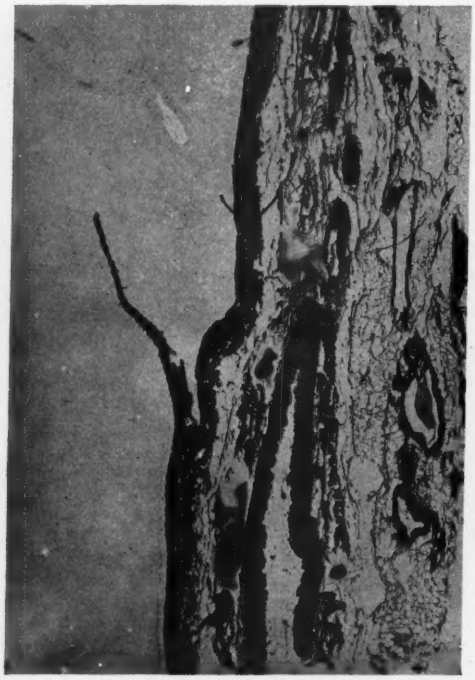

Fig. 6. Section through the cusp of well-developed valve found in the superior hemorrhoidal vein of an adult. (× 25.)

The portal system of man shows no gross valves on dissection in either the main trunk or its larger tributaries. There were only six competent valves found in one body injected with a thick barium sulfate suspension. These were in the gastric, the iliocolic and left colic regions. In one body examined there were marks left from the atrophy of a set of valves in the superior hemorrhoidal vein at the junction of this vein with the inferior mesenteric. In one specimen removed at autopsy there was a competent bicuspid valve found when the vein was opened with scissors. This segment was removed and a section made through the leaflet of the valve. (See Fig. 6.) The body from which this vein was removed was not examined at autopsy for hemorrhoids but the hospital record made no mention of finding hemorrhoids on physical examination. No other valves were found in the superior hemorrhoidal veins examined.

Injection of the portal system in adults showed the same features noted in the case of the newborn. The upper portion of the

Fig. 7. Retrograde injection of the superior hemorrhoidal vein in an adult body. Note the filling of the network of veins in the submucosa in the lower rectum and in the filling of the middle hemorrhoidal veins. The inferior hemorrhoidal veins are hardly visible.

system filled first, as did the spleen and pancreas. The larger trunks filled readily and there were no valves shown in the main tributaries after injection. The injections made with little pressure did not fill the superior hemorrhoidal vein satisfactorily. It was injected directly and the tributaries could be made out. In those injected with considerable force the injection mass showed a fine network of veins at the anorectal region as well as two or three main trunks from this area. In the bodies with no gross evidence of hemorrhoids there was only slight filling of the caval veins from the superior hemorrhoidal vein. In one body, where there were definitely visible externo-internal hemorrhoids, the injection of the superior hemorrhoidal vein filled the plexus in the rectum

and then extended into the pelvic veins on both sides. This was first noted on the subsequent *x*-ray examination and again when the thighs were disarticulated. This had taken place through the middle hemorrhoidal veins as there were only a few fine shadows at the anal margin. (Fig. 7.)

Examination of the submucosal veins in the lower half of the rectum showed that there were large veins into which were drained the much smaller veins from the anorectal region. These occurred in the region of the lower valve of Huston. In two of the adult bodies examined these veins were tortuous and ran in a direction at right angles to the lumen of the rectum. These veins were adherent to the mucosa when this was dissected from the muscular coat of the rectum and the many small veins that drained the muscularis into the submucous veins were divided. Figure 8 shows a portion of the removed anal skin and the rectal mucosa to show the location of the larger collecting veins that drain both the interior and the exterior of the rectum. The large veins present in the region so far above the hemorrhoidal area seemed to be the chief site of communication between the superior and middle hemorrhoidal veins. As they were so much larger than the veins in and around the anal canal, it seemed that in cases of portal obstruction these veins should be larger than the frequently noted hemorrhoids in the anorectal region. Examination of two cases of portal cirrhosis, one with gross externo-internal hemorrhoids and the other without, showed these enlarged tortuous and typically varicose veins. A similar case had been examined in the gastroenterology clinic some years before. At that time the examiner made the note that there were hemorrhoids present but that there were markedly enlarged veins visible in the lower 3 inches of the rectum.

DISCUSSION

This small series seems to show that there is a progressive atrophy with increasing age of valves in the portal system, comparable to that described for the other veins of the body. The increase of the width of the veins themselves seems to parallel

Fig. 8. The venous tributaries of the superior and the middle hemorrhoidal veins located in the rectal submucosa viewed by transmitted light.

the degeneration of the valves. This increase appears greater than would be expected from the growth of the body. The increase in caliber is associated with the increase of the anastomoses between the portal and caval systems around the rectum. This process may be similar in all persons, but it is possible that there is in certain families a definite hereditary weakness of the hemorrhoidal vein walls as suggested by Curtius[8] and Gutman.[14] The normal process would explain the fact that hemorrhoids are rare before puberty and why the incidence increases with age.

SUMMARY

1. The dog shows both competent and incompetent valves in the portal venous system. Two of the three bodies examined had competent valves in the superior hemorrhoidal vein.

2. The hemorrhoidal plexus in the macaque rhesus monkey resembles that in the human. No valves were found in the larger tributaries of the portal system.

3. The newborn infant has valves in the smaller portal tributaries of the gastric and colic regions. The sites of atrophic valves were noted at the mouths of the superior hemorrhoidal veins. The middle and inferior hemorrhoidal veins were protected by competent valves. The degree of anastomosis between the caval and portal systems is slight.

4. The adult has less numerous valves in the portal system. In one body there was a well developed valve in the superior hemorrhoidal vein. The central ends of the middle and inferior hemorrhoidal veins were protected by competent valves.

5. The degree of anastomosis between the portal and caval systems increases with age and is more marked in bodies showing well developed hemorrhoids.

6. The chief site of anastomosis is between the superior and middle hemorrhoidal veins and occurs in the rectal submucosa well above the anorectal line.

7. In cases of portal obstruction varix formation occurs in these veins in the rectal mucosa and these varices are visible on proctoscopic examination.

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POLYCYSTIC KIDNEY DISEASE*

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POLYCYSTIC kidney disease is of interest because of its rarity and because it is frequently overlooked. However, in the autopsy room it is striking and spectacular.[1] I have personally studied five cases, two of which came to necropsy.

In Harlem Hospital there were 67,000 admissions during the years 1930–1935, and only five cases came to light, a ratio of 1:13,400. Statistics, however, show that in 23,900 autopsies, sixty-seven cases of bilateral polycystic kidney were found. In Dr. Gordon B. Oppenheimer's[2] series at Mt. Sinai Hospital there were sixty proved cases in 220,000 admissions, a ratio of 1:3500; in 6000 necropsies fourteen cases were encountered, or a ratio of 1:428. The Mayo Clinic had an incidence of 1:3,523 in 680,000 registrations, and of 1:1,019 in 9,171 autopsies.

Bugbee and Wollstein[3] found four unilateral and eleven bilateral cases in 4,903 necropsies in infants. Ritchie[4] found two in eighty-eight cases autopsied.

Simple palpation at operation alone cannot be relied upon for proof that the non-nephrectomized kidney is normal, despite clinical reports to the contrary. It is an important surgical fact that when nephrectomy has been done for polycystic kidney in a case in which the second kidney had appeared normal to the surgeon's palpating hand, the latter kidney is very likely to become polycystic subsequent to the operation. Braasch and Schacht[5] state that polycystic kidney is always bilateral in the adult.

It is my impression that there are many more cases of this disease than our statistics show. Some cases never come under our observation because of lack of symptoms and death occurs due to some other cause; others are overlooked because they may have symptoms other than urologic. Associated complications may mask the entire picture. The clinical symptoms may be entirely those of nephrosclerosis and hypertension.

In one family under my observation, the disease occurred in three sisters, one brother and one nephew. They called themselves the "kidney family." The symptoms were all of the chronic interstitial type. In two cases the kidneys were clinically cystoscopically and roentgenologically polycystic. Two died from cerebral hemorrhages. The nephew, 45 years old, has hypertension.

Thorington[6] also has cited five cases in one family, all of the same parentage. These patients died between the ages of 30 and 50.

The disease probably follows Mendel's law of inheritance. It may occur in successive generations; only a single member of some family may have it, while in other families several may have it.

The most generally accepted theory of etiology is predicated on the assumption that the secretory and collecting portions of the kidney, developed from two different layers of the Wolffian system, subsequently unite to form a channel. A failure to join would then be responsible for the cysts. Cysts found in other organs, in my opinion, are associated manifestations.

Virchow thought that obstructions of the tubules due to uric acid and lime deposits or to inflammatory changes at the papillae led to cystic development. Shattock believed that the cysts developed from cells of the Wolffian body remnants. However, the most generally accepted theory of imperfect development is that

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founded in the dualistic theory of renal genesis.

Recently Beeson[7] has attempted to explain this failure of union as the result of a relative poverty of secretory nephrogenic tissue and atypical growth of the more abundant tubular collecting and vascular elements. Davis[8] assumes that polycystic kidney results from a delay in development and differentiation with subsequent cystic changes caused by an inherited defective protoplasm.

The adult form results from persistence of fetal maldevelopment with interstitial nephritis, and is related to granular cystic kidney. A kidney may be considered polycystic when there is an excessive number of small and large cysts throughout the parenchyma and projecting on the surface. Both cortex and medulla are involved. The cysts do not communicate with the pelves, although on occasion one may rupture into it. Arteries of varying size line the walls of the cysts, separated by a thin layer. Their rupture causes hemorrhages and resulting attacks of pain, and hematuria. Perirenal hematomas have followed rupture on the surface. A large amount of fairly normal parenchyma may remain or there may be a complete gross absence of renal tissue, depending on the stage reached.

Clinical Classification. There are two forms of polycystic kidney, the newborn and the adult. The former, more often seen in maternity hospitals, is frequently associated with various other congenital anomalies, such as hypospadias, cleft palate, harelip, spina bifida, etc. While anomalies occasionally occur in adults, they are not common.

In many of the newborn cases death occurs at birth or shortly after. In the rest the condition goes unrecognized and the patients reach adult life before presenting any signs. A few infants live to develop symptoms or die of intercurrent disease before the age of 2 or 3 years. In such instances, the syndrome of renal rickets in renal dwarfism may be present.

In the group here described the ages ranged from 28 to 44 years. Four were females and one male. They were all bilateral cases. In three cases associated or superimposed tuberculosis could not be ruled out, but radiographically polycystic disease was diagnosed.

Braasch and Schacht[5] and others have commented on the frequency of significant persistent hypertension in these cases. They attribute the hypertension to a generalized vascular disturbance as shown by the high incidence of retinal sclerosis and to the findings of obliterative changes in the arterioles and small arteries of the kidneys. Whether the vascular sclerosis, especially in the kidneys, is the primary factor and the hypertension and renal functional disturbance secondary, is a moot point. In our series, three cases had hypertension and associated symptoms, such as paralysis of the upper extremity.

Kidney Reserve. Experiments in animals, producing varying reduction of the total renal substance, while not conclusive, have shown that where a chronic state of renal insufficiency is produced, hypertension and cardiac hypertrophy result, without vascular sclerosis. Goldblatt,[9] in his recent publication on experimental hypertension due to renal ischemia, states that this type of hypertension can be produced at will by constricting both main renal arteries by means of a special clamp or by constricting the main artery of one kidney and removing the other kidney. After several years, the small arteries and arterioles show some thickening of the media and sometimes slight hyalinization of the intima, especially in retinal arterioles. This type is similar to the benign phase of human essential hypertension.

In the early stages of polycystic disease, renal impairment is not clinically evident and is found only by urologic studies of the concentrating powers of the kidney, phenolsulfonephthalin and blood chemistry tests. Patients with polycystic kidneys can

go about with marked azotemia yet without pronounced clinical signs and in apparent good health. It has been amazing to note in such patients an extremely high blood nitrogen figure and hypertension for several years before uremia occurs. This is accounted for in part by the good kidney reserve.

The hemoglobin falls because red blood cells are excreted in the urine in large quantities.

X-Ray Studies. Retrograde pyelograms are preferred with non-irritant solutions such as hippuran 40 per cent, and neoiopex 30 per cent. Intravenous pyelography is not advisable, especially where there is high blood chemistry. There is usually poor excretion of the dye because of impaired kidney function. This may further embarrass an already impaired kidney and cause an additional toxemia due to retained iodides. The pyelogram characteristically shows: (1) shortening or obliteration of one or more of the calices, producing an oval or irregular squared pelvic contour; (2) encroachment with compression of one or more calices by cysts, causing them to assume a circular or semicircular outline and change in position of the axis of the pelves; (3) stretched-out, spidery shape with sharp lines instead of the soft, curving lines of the normal contour; (4) obliteration of the pelves as the result of extension or compression of cysts. The kidneys are usually enlarged and irregular.

Diagnosis. Typical cases present a history of loin or abdominal pain, hematuria, nocturia and loss in weight. Unilateral or bilateral loin masses are noted, together with hypertension and laboratory evidence of renal impairment. A familial history is corroborative. A constant positive Mosenthal is usually present. The pyelogram is decisive.

Three types of infection are seen. In one type there is pyelitis, pyelonephritis or infected hydronephrosis, any of which may be secondary to moderate obstruction at the pyeloureteral junction by the pressure of the cysts. There may be associated calculi. The second type shows purulent infection localized to individual cysts. The third type is a diffuse purulent infection of the residual renal parenchyma, as well as of the cysts, with occasional complicating perforation and perinephritic abscess.

CASE REPORTS

Case 1. F. B., female, age 31 years, colored, was admitted December 1, 1935 with a diagnosis of pulmonary tuberculosis, polycystic kidney disease and ventral hernia.

In March, 1934 the patient complained of knife-like pains under the right scapula, later involving the anterior part of the chest. The pains were increased on deep breathing. She was acutely ill with a temperature of 104, pulse of 116 and respiration 26. Blood pressure was 110/60. A provisional diagnosis of lobar pneumonia and acute fibrinous pleurisy was made.

On March 16, the patient coughed up dark red, thick, foul sputum. Physical signs of abscess cavity were present and a tuberculosis test was positive. Bilateral tender masses in the flanks were palpable three days later. Kidney pathology was suspected, but the patient was too ill for urologic study. On April 26, operation was done for abscess in the right upper lobe of lung. The blood count was 30,000 leucocytes, with 74 polynuclears and 23 lymphocytes.

On December 3, 1934, the patient was readmitted to the hospital with complaints of cough and pains in the right side of the chest, radiating to the back. No genitourinary symptoms were noted. The thoracotomy wound was still draining, and there were pains in the right side of the abdomen, more marked in the right upper quadrant. Our impression was that there was a subdiaphragmatic metastatic abscess, possibly cholecystitis or kidney infection.

On December 19, 1934 a cystoscopy was done and catheterization of ureters. The cystoscope was passed with moderate difficulty due to a Bartholin gland abscess. The vault of the bladder was flattened due to pressure, the trigone appeared raised, and trabeculae were present in the base. The ureteral orifices were closely set, but normal in appearance. The left catheter passed up to 30 cm., the right to 20 cm. Cloudy urine was obtained from the left side and later red blood cells appeared. There was no flow from the right after a wait of twenty minutes. After intravenous injection

of 5 c.c. of indigo carmine, no dye came from the right; on the left dye appeared in twenty-four minutes, but of poor concentration.

Fig. 1. Case 1. Pyelogram, showing bilateral enlargement of both kidneys, elongated pelves, and marked distortion due to compression by the cysts.

Urine from the left side showed pus clumps, many white and red blood corpuscles, epithelial cells. Albumin was one plus. Escherichia coli were present.

Subsequent cystoscopic studies showed many pus and red blood cells, and also epithelial cells from both sides and finely granular casts.

On December 14, 1935, an intravenous pyelogram showed the kidneys enlarged. Delayed excretion of dye, incomplete filling of the pelvis and calyces in both sides were noted. A retrograde pyelogram showd elongation and flattening of the calyces and pelvis. Against these structures were seen rounded defects, the classical appearance of polycystic kidney disease.

Result of the Kahn test was two plus. Creatinine was 1.4 mg., urea 11.4 mg., and sugar 79 mg. A blood count taken on January 9, 1935 showed red blood cells 2,370,000, and hemoglobin 55 per cent.

Autopsy Findings. The liver edges were rounded and the organ enlarged, weighing 2,280 Gm.; its capsule was thin and smooth. Section of the parenchyma revealed a mosaic showing dark centers surrounded by thin rings of lighter colored parenchyma. These apparently marked out the hepatic lobules. The uterus was normal in size, and had two pedunculated fibrous tumor masses attached to fundus. The left ovary had a small cyst filled with semi-solid hemorrhagic material. The kidneys were imbedded in scant perirenal fat. They were enlarged to a combined weight of 1100 Gm. The architectural structure of the kidney was so changed that it consisted almost entirely of thin walled cysts containing fluid of various colors ranging from straw to reddish brown. A cut section of the kidney revealed complete loss of normal architecture with replacement of most of the parenchyma by cysts similar to those seen on surface. The pelves were compressed by neighboring cysts. The ureters were normal. The adrenals were elongated and narrow, apparently due to pressure of the kidneys.

Case 11. J. W., 28 years old, colored, was admitted July 11, 1933 with a chief complaint of pain in the right side for the previous two to three years. He had had pain in the right side, from behind forward, from the region of the right kidney down to the right lower quadrant. Some pains in the left side had developed in the last year. In June, 1933 acute pains in the right side had been associated with nausea and vomiting. Hematuria, polyuria, nocturia and dysuria were also reported. The patient had lues and gonorrhea.

Masses were noted in the right and left upper quadrants, moving with respiration, ballotable and tender, extending from the costal edge to the umbilicus on the right side. Right costal vertebral tenderness was present.

Phenolsulfonephthalein excretion was 20 per cent the first hour, 40 per cent the second hour. The red blood cells numbered 3,700,000; hemoglobin was 75 per cent; the white blood cells were 9,100, with polymorphonuclears 74 per cent, and lymphocyte 26 per cent.

At cystoscopy, 2 ounces of blood were obtained in urine from the bladder. Moderate trigonitis was present. Both ureteral orifices and the bladder mucosa were normal. Blood was seen from the right ureteral orifice. The catheter passed up both pelves, there was no retention, and slight bloody urine was obtained from both sides. Few leucocytes and epithelia were present in urine from the left kidney, few leucocytes and many epithelia on the right. There were 15 red blood cells per high power field, a few white blood cells, and occasional lymphocytes. Albumin was one plus.

The temperature was 99°F., the blood pressure 160/120. The blood Kahn was three plus. Blood chemistry: creatinine 1.2 mg. per cent; urea nitrogen 9.7; sugar 95.

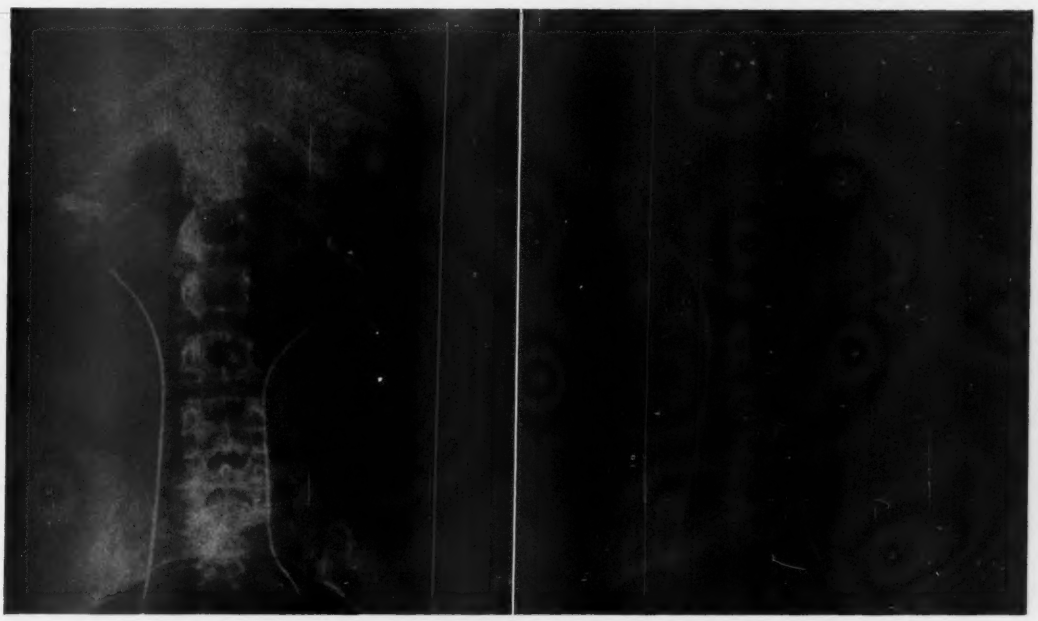

Fig. 2. Pyelogram, showing enlargement of both kidneys and elongation of both pelves and narrowing of pelvis due to compression by the cysts.

X-ray report of the urinary tract showed no evidence of stone. The right kidney appeared very much enlarged, while the left was not clearly defined. A retrograde pyelogram showed marked distortion and elongation of the right pelvis. On the left side, the pelvis was compressed from all sides. A diagnosis of polycystic kidney was made.

Case III. M. K., 44, white, was admitted on February 16, 1935 with a diagnosis of polycystic kidneys, congenital essential hypertension, and right hemiparesis. The chief complaint was weakness of the right arm and leg of two days' duration. No dizziness and no unconsciousness had occurred.

Bilateral polycystic masses were felt in both upper quadrants. The patient had a midline scar of some previous operation.

X-ray examination of the urinary tract showed enlargement of the kidneys, especially of the left. The bilateral enlargement confirmed the clinical diagnosis of polycystic kidney disease.

The Kahn test was reported negative. Creatinine was 6.0; urea 45.8; sugar 89.0. A blood count taken on February 16, showed: white blood cells 8,070, with polymorphonuclears 72 per cent, transitionals 3 per cent; lymphocytes 25 per cent; red blood cells 3,900,000, hemoglobin 70 per cent.

The patient was too ill for intravenous or retrograde pyelography and was discharged to a convalescent home on March 12, 1935.

Case IV. E. D., age 32 years, colored, was admitted on November 1, 1935 with a complaint of hermaturia. Painless hematuria had begun four years before, but cleared up spontaneously after several weeks rest in bed. In August, 1935, backaches, abdominal pains and hematuria occurred, but cleared up in a short time. The onset of the present attack began with a pelvic inflammation, bilateral pains in the kidney region with hematuria which persisted for three weeks. The patient had lost weight during the preceding few months. He denied cough, fever or night sweats. The abdominal pains had disappeared and the only symptoms remaining were hematuria, urgency and frequency.

The abdomen was soft, no masses were felt, but there was tenderness in both kidney regions. The temperature was normal.

The urine was bloody, with a specific gravity of 1.010 and one plus albumin. Microscopic examination showed many red blood cells and occasional white blood cells. The blood count was: red cells 3,600,000, hemoglobin 60 per

cent; leucocytes 10,000, polynuclears 72 per cent, lymphocytes 28 per cent.

Cystoscopy showed the bladder mucosa pale throughout, with small free blood clots floating about, and both ureteral orifices normal. There were no ulcers in bladder. A No. 6 catheter passed easily on the right; on the left side it was obstructed at 9 cm., but by manipulation passed up all the way to the pelvis. Urine from the right side was clear, from the left bloody.

Indigo carmine appeared on the right side in five minutes; on the left a faint color appeared in eight minutes.

A pyelogram showed moderate dilatation of the pelvis and calyces. There was a suggestion of multiple compression defects on both sides. Together with the history of recent hemorrhage and clear, pale urine these findings were suggestive of polycystic kidney. No acid-fast organisms were found.

Case v. A. L., age 44 years, white, was admitted on September 26, 1933 with a complaint of hematuria and pains in both flanks. Four days prior to admission the patient had noted spontaneous, painless bleeding through the urethra and the need for frequent micturition. On September 24 nausea and cold sweats occurred and later the patient vomited and had pains in the left, lower quadrant. Insomnia and restlessness were present. A renal calculus had been passed twenty years before and gonorrhea had been contracted three years before.

Moderate ascites and tenderness were present over the entire left abdomen, a mass was palpable on the left, filling the abdomen from the costal margin to three to four fingers below the umbilicus. The masses were hard, tender, irregular, nodular and not movable. There was left costal vertebral tenderness; the right side of the abdomen was normal.

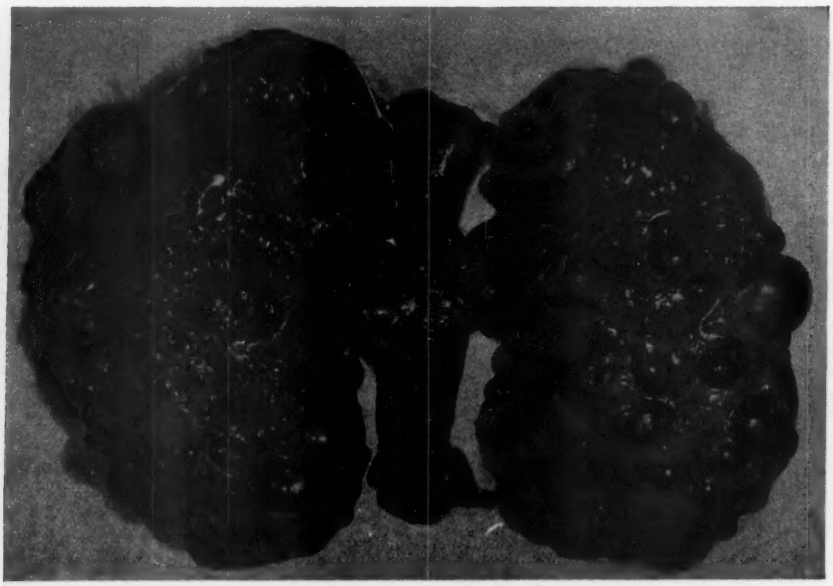

Fig. 3. Case v. Gross enlargement of both kidneys. Many cysts of various sizes. Patient was too ill for pyelogram.

Blood chemistry showed creatinine 13.9 mg. per cent, urea 116.2, sugar 125. The Kahn was negative. Blood pressure was 160/100, temperature before operation 102°F. and later 104. The white blood cells were 25,000, with polynuclears 88 per cent. Hemoglobin was 65 per cent.

Cystoscopy produced bloody urine from the left side, clear on the right. Indigo carmine appeared late and in poor concentration from both sides.

Operation was done on September 26, 1933 with the idea that a perinephric abscess was present. A left flank incision exposed a large polycystic kidney. Puncture of the cyst caused purulent and sanguinous fluid to exude. A drain was inserted.

At autopsy the pericardium contained about 10 c.c. of straw colored serous fluid. The heart was of average size. The musculature of the left ventricle showed thickening. The aorta and coronaries showed some fatty deposition.

Where the abdomen was opened, there was a small amount of hemorrhagic fluid in the lateral

gutters. The spleen was of average size, its borders well defined. There was a large lobulation which extended into the organ of about ¾ inch. On section, the cut surfaces were darkened. The liver was of average size, its capsule smooth. The quadrate lobe presented small cysts of various sizes from a pinhead to a pea. The kidneys presented two large masses much greater than average. On the left there was marked extravasation of blood into the perirenal tissue. The surface of the kidney consisted of a series of cysts of various sizes and colors, sized from a pea to a marble. The right kidney presented a similar appearance. The left ureter was dilated.

The autopsy diagnosis was uremia; bilateral polycystic kidney; atherosclerosis; cystic liver.

COMMENT

Treatment recommended is operation, puncture of cysts, and decapsulation. Nephrectomy should never be done except possibly in cases of persistent suppuration in which other surgical measures have failed and in which death may result from continued infection.

Medical treatment is that given in any chronic nephropathy, including rest, avoidance of physical or mental stress, or exposure to infection and dietetic regulations to prevent any overloading or irritation of the diseased kidney. When pyelitis or pyelonephritis complicates the condition, catheter drainage and pelvic lavage with a weak solution of silver nitrate may be tried. Calculus complications may require surgical interference, if a stone blocks the ureter or the pyleureteral junction and conservative cystoscopic measures have failed. Surgery should not be done in other stone cases.

I wish to thank Dr. Solomon Weintraub, pathologist and Dr. William Snow, roentgenologist, for their respective opinions.

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CASE REPORTS

HETEROTOPIC GASTRIC MUCOSA AND REDUPLICATIONS OF THE INTESTINAL TRACT*

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WHILE the occurrence of gastric mucosa in ectopic foci throughout the body may be responsible for severe clinical signs and symptoms and may even lead directly to the death of the patient, it is also true that proper surgical treatment may produce a complete cure. In view of these facts we should like to call attention to the occurrence of this type of congenital abnormality in various portions of the gastrointestinal tract and related structures and report three unusual cases which nevertheless illustrate well its more common pathologic effects.

The presence of islands of gastric mucosa in the esophagus and in remnants of the vitelline duct (Meckel's diverticulum) is common. Nicholson[1] states that there is an incidence as high as 75 per cent in the upper portion of the esophagus. In a report of thirty-two cases of Meckel's diverticulum, Hudson and Koplik[2] found gastric mucosa in 52 per cent. This corresponds very closely with the figures from the surgical and autopsy records of the Mallory Institute of Pathology where, of the last twenty Meckel's diverticula examined histologically, ten, or 50 per cent, showed islands of ectopic gastric mucosa. Aside from these two locations the occurrence of ectopic gastric mucosa is extremely rare. It has, however, been reported in the duodenum by Taylor,[3] in the jejunum by Barták[4] and by Kimpton and Crane,[5] in the ileum by Poindecker[6] and by Taylor,[3] and in the pancreas, gall-bladder and colon by Nicholson.[1] Its occurrence in the umbilicus in remnants of the vitelline duct has been described by Stone[7] and by Nicholson.[8]

Islands of ectopic gastric mucosa produce no typical or pathognomonic clinical signs or symptoms, these depending to a large extent upon the site of their occurrence. However, the one sign which they may have in common, regardless of their location, is hemorrhage into the gastrointestinal tract. The cause of the bleeding is the frequent presence of typical peptic ulcers either adjacent to or involving the ectopic mucosa, with ulceration of blood vessels which may actually lead to death from exsanguination. The importance of ulceration and hemorrhage in connection with Meckel's diverticula has been emphasized by previous writers[2,9,10] and Hudson[11] states that 31 per cent of Meckel's diverticula may show hemorrhage on this basis. In considering the various possible causes of intestinal hemorrhage this condition should be remembered, particularly when dealing with intestinal hemorrhage in children. The ulcerations associated with this anomaly may also lead to perforation with peritonitis or mediastinitis. The heterotopic mucosa may be of sufficient size or may proliferate to sufficient size to produce the clinical picture of a tumor with a palpable mass, obstruction, volvulus or intussuscep-

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tion. The following three unusual cases will serve to illustrate some of these possible dangers of heterotopic gastric mucosa.

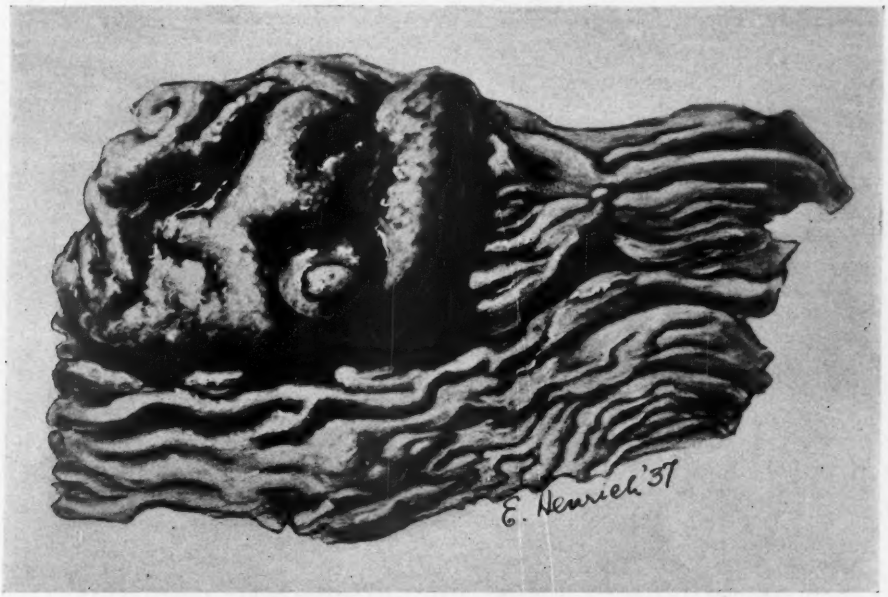

Fig. 1. Case 1. Gross specimen showing tumor nodule in jejunum. The gross resemblance of the mucosal surface of the tumor nodule to gastric mucosa is striking. About two times actual size.

CASE REPORTS

Case 1. J. B., a 7 year old girl, was seen in consultation by one of us (A. R. K.) because of recurrent attacks of severe paraumbilical pain associated with nausea and vomiting since the age of 3. She was born at full term after a normal pregnancy, the fifth child of a family of nine. Her weight at birth was 8¾ pounds and no physical abnormalities were noted. Her parents and siblings were entirely normal, and there were no pertinent factors in her past history other than those associated with her present illness.

The first abdominal complaint occurred at the age of 3 and was manifested by severe pain about the umbilicus, not localized, and marked by a continuous ache with occasional attacks of excruciating pain causing her to cry out. After two or three hours the child vomited and the pain was relieved. Similar attacks occurred subsequently three to six times a year. They lasted from one to three days, and began during the night as frequently as during the day. Following each attack the child appeared perfectly normal except for a sense of weakness. Fever never developed during any of the attacks. The mother always gave the child a laxative; this was invariably followed by a watery defecation, but at no time were the stools abnormally dark or bloody. The attacks gradually became more frequent and severe, but no definite inducing factor was ever noted. Two years after the onset of this condition a physician was consulted for the first time and a diagnosis of intestinal worms was made. Three weeks later a second physician was consulted and for the first time an abdominal mass was palpated; the mother stated that a mass could be seen especially when the pain was severe. At this time a diagnosis of volvulus was made, the abdomen was tightly strapped with tape, and abdominal massage and shaking the child in an upside-down position by holding the feet were advised. Following this the attacks were much less frequent. One year later, because of a severe attack, the patient was referred to a hospital, where *x*-rays of the intestinal tract revealed nothing abnormal. During the year preceding the present examination she had had attacks of varying severity every four or five weeks. She was seen in consultation on June 21, 1937, having just suffered an acute attack of abdominal pain.

Physical examination was negative save for the abdomen, where an elongated tumor could be felt extending diagonally across the abdomen from left to right. It was 10 or 12 cm. in length, 2 or 3 cm. in width, soft, smooth and non-

tender. The center of the mass lay just beneath the umbilicus. The appearance was quite typical of intussusception. While lying quietly on the examining table, the child suddenly screamed, doubled up, holding her abdomen, became pallid and perspired profusely. This lasted for about ten minutes and was followed in a few minutes by a similar attack. The abdominal tumor became very prominent through the child's thin abdominal wall while the pain was most severe.

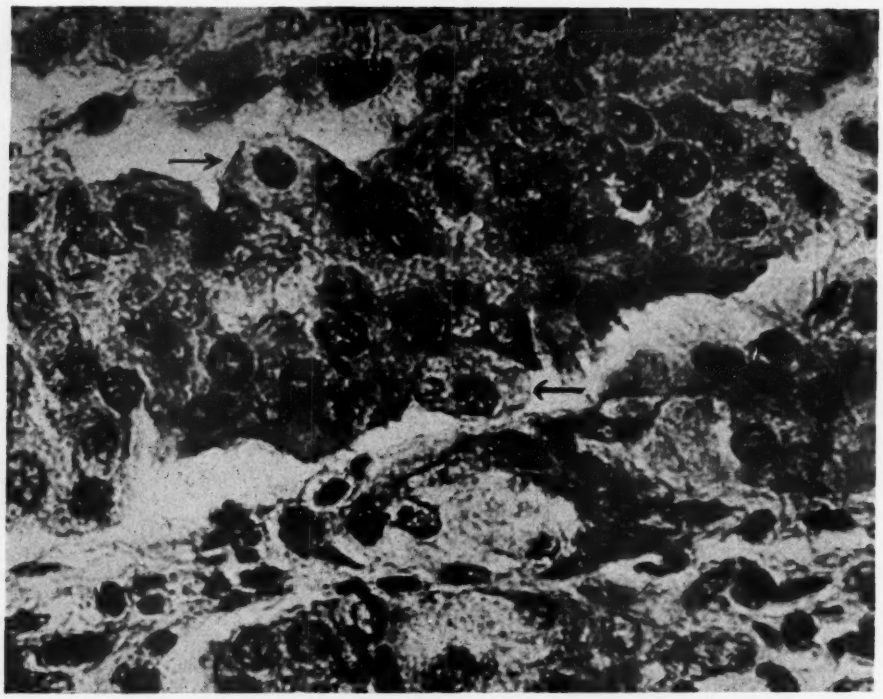

FIG. 2. Case 1. High-power field of the tumor mucosal cells, showing large, round parietal cells (indicated by arrows). Hematoxylin and eosin. × 910.

At operation, the abdomen was exposed by a left paraumbilical midrectus incision and a large intussusception of the jejunum was found starting about 15 cm. below the ligament of Treitz. The bowel was edematous but entirely viable, and the intussusception was reduced with ease. When it was completely reduced, a soft, dough-like tumor mass was found filling the entire lumen of the jejunum 20 cm. below the ligament of Treitz. This was resected without difficulty by removing 12 cm. of jejunum, and a side-to-side anastomosis was done. The abdomen was closed in layers without drainage.

The immediate postoperative course was uneventful. Eighteen months after the operation the child was healthy and had had no recurrence of her previous symptoms.

Pathologic Report. The gross specimen consisted of a piece of small intestine (jejunum) measuring 7 by 5 by 0.4 cm. The serosa was negative but the entire wall was thickened and edematous, although not otherwise remarkable. The mucosa in one area measuring 3 cm. in diameter was redundant, being elevated 0.4 cm. above the surrounding tissue to form a definite tumor nodule with a broad base. There was no evidence of infiltration of the underlying tissues.

Histologic sections through the redundant portion of mucosa showed it to be composed of long mucous glands of the gastric type in some of which numerous large, round, eosinophilic parietal cells could be readily identified. At the margin of the nodule there was an abrupt transition to a mucosa of the jejunal type, with many large plicae circulares and numerous goblet cells. The tumor nodule itself lay entirely on the mucosal side of the muscularis mucosa, and the redundancy of the mucosa noted in gross examination was due to the numerous gastric glands of which the tumor was composed. There were occasional mitotic figures in the epithelium, but the cells were perfectly differentiated into adult chief and parietal cells, as found in the mucosa of the stomach. The submucosa, muscularis and serosa were normal save for evident intercellular edema.

This case is essentially a typical one of intussusception, but is unusual and of interest because of the embryologic anomaly which was responsible for the intussusception. The rôle of tumors in the production of intussusception, particularly in the adult, has been emphasized by recent investigators,[12,13,14] although the association of the two lesions has long been recognized. Roan,[13] in reporting a series of cases of intussusception, states that tumors are a negligible factor in children up to the age of 3. Since benign tumors are much commoner than malignant ones in the small intestine, they predominate in any analysis of tumors causing intussusception. Nevertheless, the polypoid nature of many benign intestinal neoplasms renders them even more potent factors in the general etiology of intussusception. In all reported series the commonest types of tumors encountered are myomas and adenomas, although instances of fibroma, lipoma, hemangioma and other benign tumors are not infrequent.

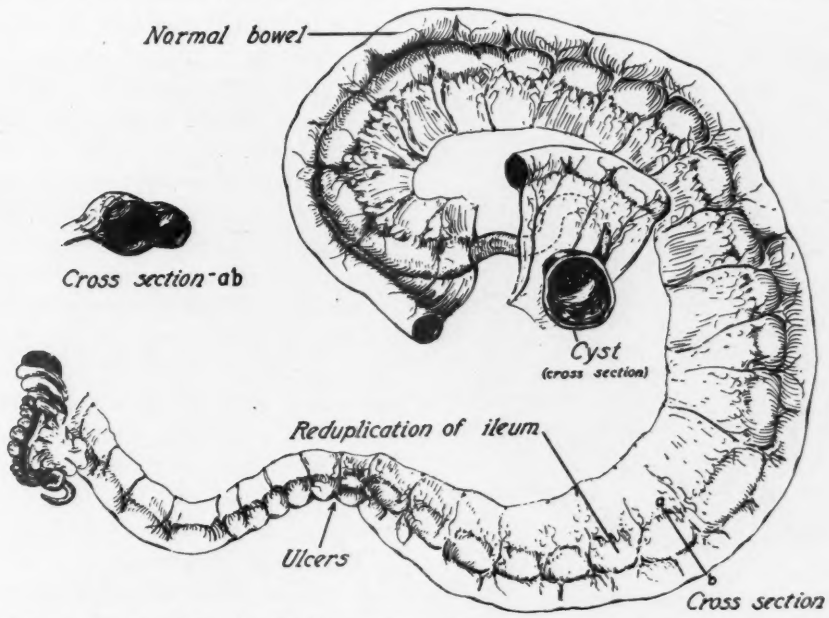

Fig. 3. Case II. Drawing to demonstrate the abnormality of reduplication of the ileum. Cecum and appendix are on left.

Case II. C. C., a 9 months old baby boy, was admitted to the Boston City Hospital because of bleeding by rectum. He was the first child of a 28 year old white housewife and was born at term. It was noted in the hospital after his birth that he vomited frequently, but his parents never returned him to the hospital for study as advised. There were no other pertinent factors in his past history other than those associated with his present illness.

The first appearance of rectal bleeding occurred at the age of 3 months about three weeks after the onset of pertussis. At that time the child had three red to tarry stools. He was admitted to the South Department of the Boston City Hospital because of the pertussis and rectal bleeding, but was taken home against advice before he could be properly studied. There had been no recurrences of the rectal bleeding up until the day of the last admission when his mother noted the presence of black tarry stools and fresh blood in his diapers twice within a period of two hours.

The physical examination showed marked pallor of the skin, a hard round mass about the size of a lemon in the left upper quadrant of the abdomen, distention of the abdomen and the presence of blood in the rectum. In addition he showed some beading of the ribs and enlargement of the head with prominent frontal bosses. The physical examination was otherwise negative. The only laboratory data gathered were a hemoglobin of 25 per cent, a red blood count of 2,060,000, and a white blood count of 17,200 with 80 per cent polynuclear cells.

A laparotomy was performed and a giant diverticulum of the ileum was found. The ileum became double-barreled at a point about

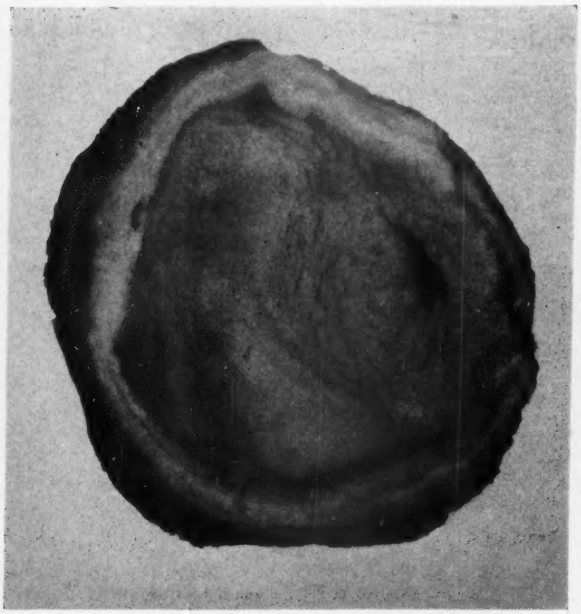

Fig. 4. Case II. Cyst after cross section showing the rugose gastric-like appearance of the lining and a typical "punched-out" peptic ulcer. About actual size.

25 cm. from the ileocecal valve. This double ileum continued proximally for about 95 cm., the inner or false bowel lying in the mesentery of the outer or true bowel but in close approximation to it. The false segment then dipped towards the base of the mesentery penetrated the mesentery and ended in a round cystic structure about 6 cm. in diameter, apparently corresponding to the mass noted clinically in the left upper quadrant of the abdomen. It was felt that the cyst and an ulcer which could be palpated at the bifurcation of the ileum were the main sources of the bleeding and therefore the cyst, about 5 cm. of the diverticulum with the cyst and about 5 cm. of ileum at the point of bifurcation were resected. A side-to-side anastomosis was then done between the terminal ileum and a portion of ileum overlying the diverticulum.

Pathologic Report. The gross specimen consisted of three parts. The first was a piece of intestine from the bifurcation of the ileum 5 by 4.2 by 0.3 cm., the surface of which was gray-red and edematous. In the middle of the mucosal surface of this fragment were three punched-out ulcers, the largest 1 cm. in diameter and 0.3 cm. in depth, the others being but slightly smaller. The other two portions of the specimen were a piece of intestine measuring 5 by 1 by 0.3 cm. and somewhat resembling an appendix and a cystic structure 6 cm. in diameter filled with partially digested black blood and showing a rugose gastric-like mucosa. It contained approximately eight punched-out ulcers, the largest measuring 1 by 1 by 0.6 cm.

Histologically all three of these segments showed typical gastric mucosa with both chief and oxyntic cells the latter being extremely abundant. The muscle layers of the segments were those of small bowel save for that of the cyst which showed interlacing muscle bundles similar to those seen in the normal stomach. The ulcers were sharply defined, in some areas extended deeply into the muscularis and showed a moderate focal lymphocytic reaction.

Following the operation the patient showed progressive elevation of temperature and pulse and died within twenty-four hours.

The only important autopsy findings were those related to the intestinal tract. The large bowel was not remarkable, and 23 cm. proximal to the ileocecal valve there was a side-to-side anastomosis between two closed and sutured ends of ileum. The ileum proximal to the anastomosis was double, being composed of two distinct tubes which were inseparably fused at their adjacent surfaces. The inner intestinal tube was greatly distended as it lay in the mesentery of the outer or normal ileum. The mesenteric vessels to the true bowel circumscribed this inner bowel. These intestinal tubes ran parallel for a distance of 90 cm. at which point the inner tube became detached and ran for a short distance towards the base of the mesentery. The outer intestine continued on proximally to join a normal jejunum, duodenum and stomach.

Histologic sections showed this inner segment of bowel to be lined throughout by gastric mucosa with numerous oxyntic cells. The muscularis was incomplete, being entirely lacking in the portion where the two tubes were adjacent although the remainder of the muscularis of each segment was normal.

Case III. F. W., a 77 year old white male, was admitted to Boston City Hospital because of edema and dyspnea of increasing severity. Because of his poor condition no reliable history was obtained. On physical examination he showed an enlarged heart, blood pressure of 140/90 and marked dependent edema. Labora-

tory data were not significant. The patient did well for a few days and then showed increasing signs of cardiac failure and died twenty days after admission.

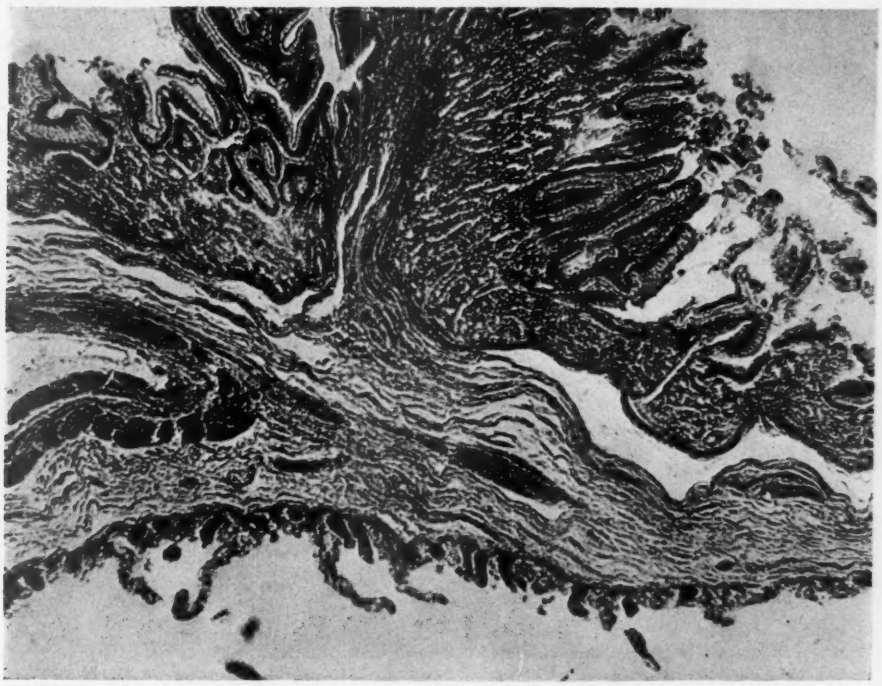

Fig. 5. Case II. Cross section through normal bowel and reduplication at point indicated in Figure 3. Gastric mucosa lining reduplication above; lumen of true bowel with normal mucosa below. Note the absence of the muscularis between the two lumens on the right of the photograph. Hematoxylin and eosin. × 30.

Autopsy showed ulceration in the esophagus about 4 cm. from the cardia and measuring about 3 cm. in diameter, with an irregular edge and a green sloughing base extending into the wall for about 0.2 cm. In addition signs of cardiac decompensation and a bilateral bronchopneumonia were apparent.

Histologically at the margin of the ulcer a few glands similar to those of the stomach could be identified. These glands showed only chief cells, however, no oxyntic cells being seen. At the base of the ulcer there was complete destruction and fibrous replacement of the normal muscularis of the esophagus, and in addition there was a marked chronic inflammatory reaction throughout the wall of the ulcer with a moderate diffuse lymphocytic and plasma cell infiltration.

These two cases serve to show the association of ulcer and bleeding with ectopic gastric mucosa. The explanation of the symptomatology of Case II is clear enough since there were bleeding ulcers in various portions of this diverticulum connecting directly with the bowel lumen. It would seem that autodigestion by the local concentration of hydrochloric acid in the sac and at the opening of the diverticulum may have been a most important factor in the development of the ulcers. It is unfortunate that a more pertinent history was not available in the third case, but Lyall[15] states that the common clinical symptoms of ulcer of the esophagus are pain, dysphagia and hematemesis. In regard to peptic ulcers of the esophagus Jackson[16] lists the following causes: focal infection, retrograde flow of gastric juice, cardiospasm, insufficiency of the cardia, insufficiency of the diaphragmatic pinchcock, and ectopic gastric mucosa. He points out that the occurrence of gastric mucosa in the esophagus may be an active factor by the secretion of gastric juice or passively by supplying a tissue that may be sensitive to concentrated gastric juice. The role of ectopic gastric mucosa in this type of ulcer of the esophagus is also emphasized by Lyall and by Hurst[17] who feel the most

important factor is the active secretion of hydrochloric acid by the ectopic tissue and the trapping of this acid in the lower portion of the esophagus by cardiospasm. The commonest complications of peptic ulcer of the esophagus are perforation and hemorrhage. The extensive damage evident in Case III shows how such ulcers may well go on to perforation and mediastinitis.

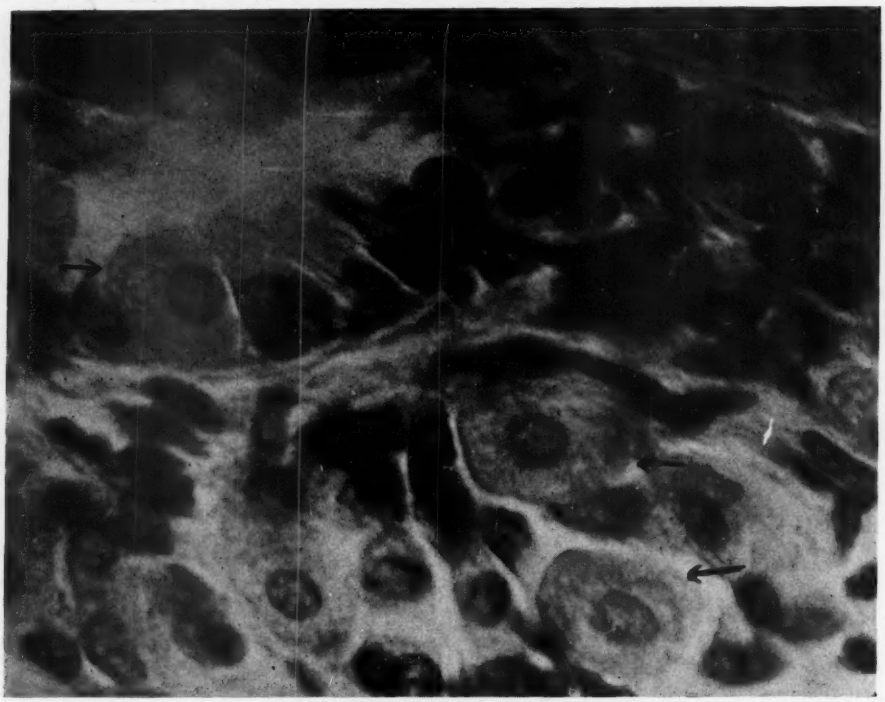

FIG. 6. Case II. High lower field of gastric mucosa shown in Figure 5. Note the large round parietal cells. Hematoxylin and eosin. × 1200.

ETIOLOGY

The reasons for the development or existence of ectopic gastric mucosa are entirely unknown although numerous theories have been suggested. We agree with Nicholson[1,8] who concludes that the developmental heteromorphoses indicate that the original prospective potencies of cells are wider than their prospective values; and that the accidental heteromorphoses suggest that the original prospective potencies are not entirely lost during development, since they are sometimes accidentally revealed in pathologic states in old age. In other words, one must bear in mind the common origin of the intestinal epithelium and realize that each cell may differentiate in various ways. This, however, carries us no closer to the pathogenesis of such lesions, beyond removing the necessity of explaining all such findings on the basis of embryologic rests. Curd[18] reviews the various theories of origin and development of ectopic gastric mucosa. Briefly stated, these are:

1. The presence of a substance in the intestines which stimulates the formation of intestinal mucosa, and the lack of which is necessary for the formation of gastric mucosa. This substance was thought to be bile.

2. An inflammatory process in the endoderm during some stage of embryologic development.

3. Misplaced fetal inclusions or rests.

4. The rapid growth of the intestines does not allow the differentiation of cells into the gastric type, while the smaller size and slower growth of the stomach allow for complete differentiation of the gastric mucosa.

The first theory has been proved incorrect and is further disproved by the first case reported here. The theory of rapid

development of the intestines is ruled out by our second case. It is impossible to conclude what mechanism governs the development of ectopic gastric mucosa in the light of our present knowledge.

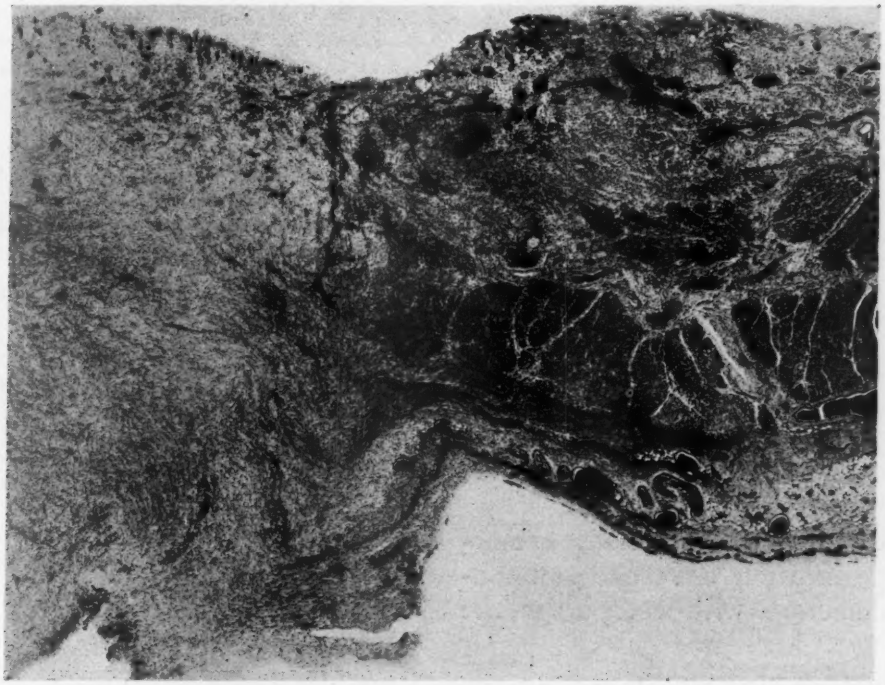

Fig. 7. Case III. Section of esophageal ulcer showing extensive scarring and complete destruction of the muscularis and marked chronic inflammatory reaction extending through to the adventitia. Mucosal surface is above. Hematoxylin and eosin. × 15.

Case II showed an unusual deformity in addition to the ectopic gastric mucosa. Hudson collected eighteen such cases from the literature and reported three of his own under the title of "Giant Diverticula or Reduplications of the Intestinal Tract." The pathogenesis of this type of anomaly is likewise entirely unknown. Hudson[19] lists the following theories:

1. Atavism or reversion to an earlier phylogenetic form.
2. Twinning, in which case "the stimulation to reduplication, although acting at an early stage of cell division has affected a certain section only of the embryo."
3. Through the development of a median septum.
4. Sequestration of a segment of the intestinal tube.
5. Persistence and alterations of the vitelline duct.
6. The nodules of Lewis and Thyng.[20] These authors noted nodules of epithelial cells occurring along the course of the gastrointestinal tract in pig, rabbit and human embryos. Normally these disappear or coalesce to form a part of the lumen of the digestive tube but they may persist.

The evidence is not conclusive for any single theory, but Hudson concludes that the evidence favors the belief that the nodules of Lewis and Thyng[20] are antecedents of those cysts and diverticula not associated with remnants of the omphalomesenteric duct.

TREATMENT

The only satisfactory treatment of islands of heterotopic gastric mucosa regardless of the location or form of their occurrence is complete surgical removal where possible. Reduplications of the intestinal tract should also be completely excised since they may be wholly or partially lined with gastric mucosa. At this point we should also like to emphasize the importance of a thorough surgical explora-

tion in individuals, and especially children, presenting vague or unusual but persistent intestinal symptoms particularly when associated with gastrointestinal hemorrhage. In the case of the esophagus Jackson[16] recommends removal of foci of infection, cauterization of ulcers with weak silver nitrate and a dietetic régime such as would be advised for a patient with a gastric ulcer of the stomach with especial attention to the avoidance of rough, harsh and bulky foods.

SUMMARY

1. Three instances of heterotopic gastric mucosa are reported (esophagus, jejunum and giant diverticulum of ileum), two of which were known to have been responsible for clinical symptoms.

2. The dangers of ulceration, hemorrhage and perforation have been emphasized in connection with ectopic gastric mucosa.

3. The theories of origin of heterotopic gastric mucosa and giant diverticula of the intestine are presented.

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PURULENT PERICARDITIS*

REPORT OF A CASE WITH UNUSUAL COMPLICATIONS

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PYOPERICARDIUM may occur as a serious complication of some concurrent disease, most often another suppurative process within the chest. The treatment is surgical and carries a mortality of about 50 per cent. Conservative treatment is of no value. This mortality rate is still higher when complicated by empyema, particularly of the left side. In view of these facts and the scarcity of reports in the literature we feel justified in presenting a case of streptococcus pneumonia with a left-sided empyema, purulent pericarditis, massive right pleural effusion, ascites, and thrombophlebitis of the right jugular vein.

N. V., a 16 year old white male, was admitted to the Wisconsin General Hospital on May 1, 1937, because of pain in the left chest. About three weeks prior to admission he had developed a "head cold," and on April 23, 1937, there had followed malaise, cough, dyspnea, and fever. A diagnosis of bilateral pneumonia had been made by his local physician. The symptoms slowly subsided, but the cough, at first non-productive, began to yield a thick, blood-streaked, odorless sputum. Fever continued in the neighborhood of 100°F. The night before admission an acute pain developed in the left chest, upper abdomen, and the shoulder of the same side and the fever rose to 105°F.

The patient was alert, dyspneic, coughing paroxysmally. There was a lag of the left hemithorax during respiration accompanied by a friction rub anteriorly, increased tactile fremitus, and dulness over the entire left chest as high as the second interspace anteriorly. Breath sounds were absent or greatly suppressed over the same area. A few crackling râles were present at the right base. The heart rate was rapid (136 per minute) with an accentuation of the pulmonic second sound. To percussion the heart was of normal size and shape. A diagnosis of left upper lobar pneumonia with massive pleural effusion was made and confirmed by *x*-ray.

Laboratory findings: Hemoglobin 14.7 Gm. Red blood count 5,350,000; white blood count 33,500, with 96 per cent neutrophiles. Nonprotein nitrogen 23 mg. per cent. The sputum was negative for pneumococci types 1 to 4, but showed a hemolytic streptococcus. Serum protein was 6.1 Gm.

The patient's course was stormy and marked by a profound toxemia. A thoracentesis performed on the third day after admission revealed a slightly cloudy fluid which on culture also showed hemolytic streptococci. Therapeutic doses of oral and intramuscular sulfanilimide were accompanied by the rapid development of cyanosis and were discontinued before any beneficial effect could be ascertained. To relieve dyspnea and await the formation of a pyogenic pleural membrane, the empyema cavity was aspirated daily until it was impossible, by this method, to keep pace with the rapid pus formation. On May 19, intercostal drainage was instituted and the pleural cavity was decompressed slowly over a period of forty-eight hours. The respirations improved with closed drainage as did the general condition, until June 16, when there appeared a new symptom—substernal pain. Bedside chest films taken at this time showed a heart shadow somewhat larger than on previous examination, but because of the extensive pleural reaction and the fluid, as well as consolidation at the left base, it was felt that a diagnosis of pericardial effusion could not be made roentgenographically.

During the next fifteen days an attempt at pericardial tapping yielded nothing and the patient became progressively worse. On July 4

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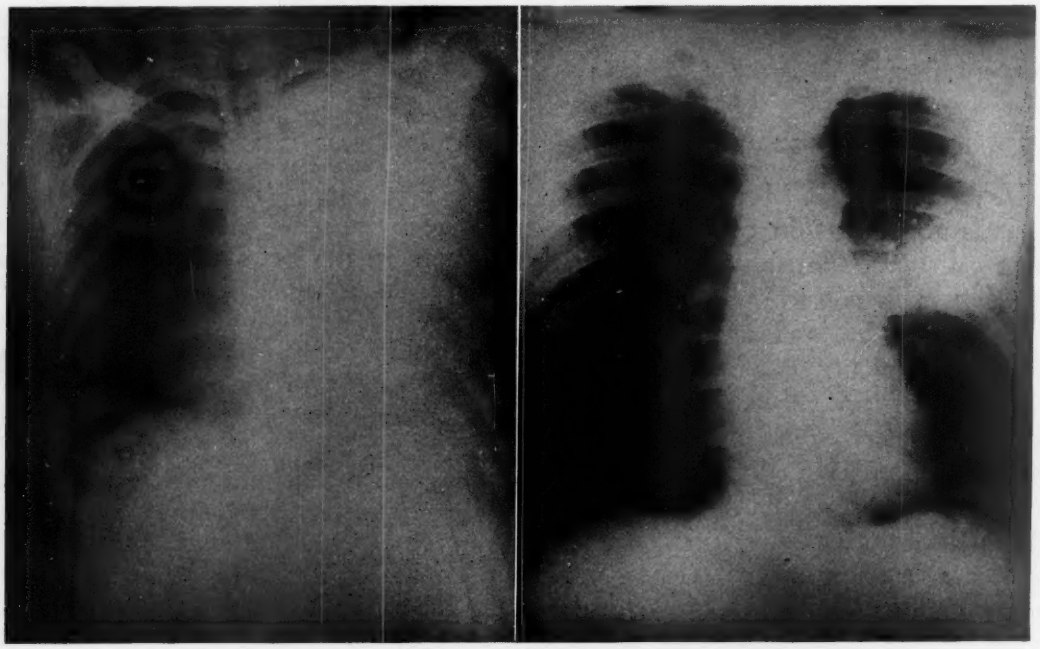
an acute circulatory collapse developed, characterized by extreme dyspnea, with cyanosis, profound shock and muffled rapid heart tones.

Further treatment consisted in the irrigation of the two sinuses, hot compresses to the neck, and frequent decompression of the right plueral



A B

FIG. 1. A, pleural effusion entirely obscuring the left lung and pushing the heart slightly to the right. B, following an intercostal thoracotomy the haze over the left lung is less dense and the heart has returned to its normal position.

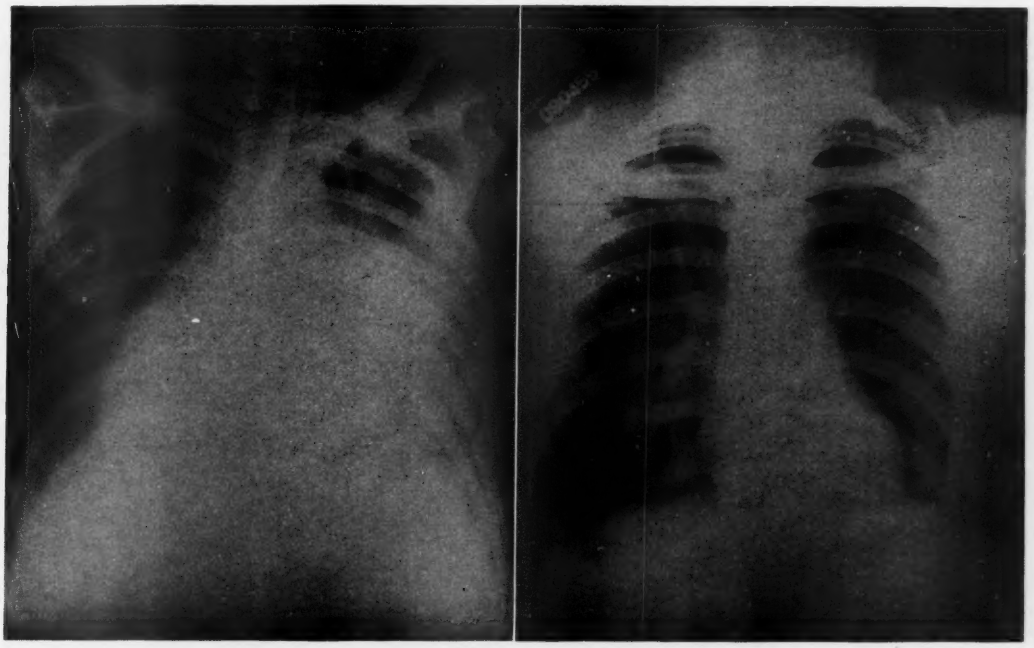
Pericardial tap was again considered advisable and yielded 100 c.c. of sanguinopurulent fluid. With the patient in extremis, pericardiotomy was performed under local anesthesia by subperichondral resection of the fourth and fifth left costal cartilages. As the sac was incised, pus under tension welled up. In all 950 c.c. was recovered. A 24 F. catheter was inserted. Improvement in breathing, color, and pulse was immediate. Cultures of this pus showed hemolytic streptococci.

As early as July 1, there were signs of fluid rapidly accumulating in the right chest. Every other day amounts ranging from 500 to 1600 c.c. of straw-colored, coagulable fluid were removed. By July 12 there was a massive collection of ascitic fluid in the abdomen and edema of the right neck with marked tenderness along the course of the internal jugular vein on that side. At this period both pericardial and left empyema sinuses were draining well but the question of possible pocketing in the former was raised. Numerous pericardial adhesions were found on gloved finger exploration and these were lysed with improvement in drainage. cavity by thoracentesis. The ascites persisted for almost a month, but abdominal paracentesis was not deemed advisable as long as adequate vital capacity was maintained by right chest aspiration. Improvement was gradual but progressive, and both sinuses were closed at the time of discharge on September 11, 1937. Since dismissal the patient has remained well.

Electrocardiographic tracings in this case are interesting. The electrocardiogram taken July 20, sixteen days after pericardiotomy and while the pericardium was draining well, showed low voltage, slight depression of the S. T. segments in all the limb leads, and inversion of the T waves. By September 9, all drainage had ceased and a second electrocardiogram then showed an increase in the amplitude of the Q.R.S. complexes in all leads, almost isoelectric S.T. segments, persistence of inversion of T_1, T_2 was tending to become positive, T_3 was definitely upright, and T_4 taken with the old technique[10] was upright. The last electrocardiogram taken almost a year after surgery showed still further increase in Q.R.S. amplitude especially in leads II and III, upright T

waves in leads I and II, diphasic T in lead III, and T_4 had become diphasic with the new technique.[10]

came the appearance of abdominal fluid which we feel could be only a transudate, although its identity was never proved.



A B

FIG. 2. A, the left lung field is obscured by a pleural effusion probably due to inadequate drainage of the empyema cavity. The cardiac shadow is definitely enlarged and of the "water bottle" type. B, June 10, 1938, following thoracotomy. Almost complete return to normal. Note obliteration of the left costophrenic sulcus.

Katz, Scott, and Feil[1,2] reported that in experimental distention of the pericardial sac there is a decrease in the Q.R.S. amplitude, elevation of the S.T. segment, and inversion of the T wave. Experiments of Bay, Gordon, and Adams[3] produced the same results except that the S.T. segments were occasionally depressed. In a later clinical article by Vander Veer and Norris[4] electrodiographic changes in fourteen cases of pericarditis were presented with inclusion of the chest lead. Six cases of this series showed elevation of the R.T. segment to be the most pronounced finding, and interestingly enough, the chest lead was normal.

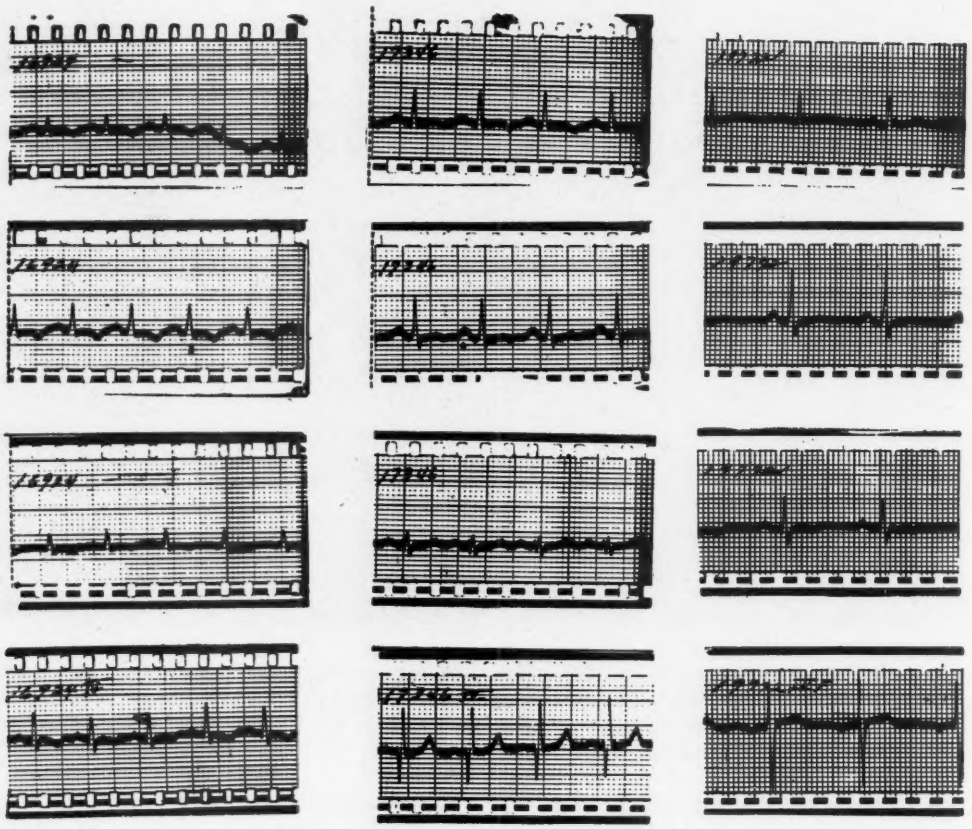
For some of the following statements we have basic proof; the rest are only hypotheses. The right-sided pleural effusion was a sterile transudate which coagulated on standing and developed at a time when the right pulmonary fields were clear and mechanical stresses within the chest were greatest. Almost simultaneous with this

There are two explanations for these accumulations; namely, intrathoracic impediment to venous return or cardiac failure. The former seems most likely in a patient of this age without previous evidence of cardiac disease. Part of this is substantiated by the presence of venous obstruction in the right side of the neck accompanied by unilateral uniform swelling, marked tenderness, and a palpable thrombosis of the external jugular vein.

It is true that dependent edema of the lower extremities did exist, and this in the absence of a hypoproteinemia is explained on a basis of intra-abdominal pressure on the iliac vessels.

The former contention of the utter futility of drainage of pyopericardium because of the frequency of complication later by obliterative pericarditis has long been exploded. Shipley and Winslow[5,6] reported only two cases of adhesive pericarditis following drainage of a pyoperi-

cardium in an extensive review of the literature up to January 1, 1935. In our case there was evidence of adherence of the muscle or the cardiac chambers. In a patient already taxed to the utmost by his disease, the latter trauma may be sufficient



A B C

Fig. 3. Electrocardiograms. A, July 20, 1937; B, September 9, 1937; C, June 10, 1938.

pericardium to the operative scar one year after operation, but certainly no evidence of any degree of obliteration of the pericardial sac or decrease in the gross cardiac reserve. They[5,6] contend that purulent pericarditis is an abscess, similar to a collection of pus anywhere else, and should be similarly treated by adequate incision and drainage. As previously stated, operative treatment yields a mortality rate of 50 per cent, but anything short of this, including aspiration at periodic intervals, is uniformly fatal.

Most writers[2,3] feel that aspiration in purulent pericarditis should not be attempted; first, because a negative tap frequently occurs and leads to a false sense of security, and secondly, the needle is apt to be inserted directly into the heart to cause death. The collection of pus is greatest behind and below the heart causing this organ to be in close proximity to the anterior pericardium and chest wall. In our own case a sanguineous fluid was obtained by aspiration, yet pericardiotomy performed immediately afterward revealed frank pus. On the other hand, aspiration in the case of serous pericarditis is fully as efficacious as pericardiotomy. Caughey[7] and Stewart[8] report satisfactory results with this procedure.

Pocketing of pus occurred behind the heart, giving the syndrome of obstruction to the vena cava. Re-exploration of the pericardial sac ten days after the pericardiotomy, breaking up adhesions and reestablishing drainage were sufficient to relieve this obstruction. Even with this

unfavorable circumstance, cure eventually resulted.

CONCLUSIONS

1. A case of streptococcal empyema complicated by a purulent pericarditis was successfully treated by drainage.

2. Early and adequate drainage of purulent pericarditis is the treatment of choice and rarely results in postoperative adhesive pericarditis.

3. Aspiration of a purulent pericarditis may be both ineffectual and dangerous.

4. Evidence of pressure on the vena cava following pericardiotomy may indicate pocketing of pus behind the heart and re-exploration of the pericardial cavity is advisable.

5. Electrocardiographic tracings are an aid in arriving at a diagnosis of pericarditis with effusion. The changes to be noted are: elevation or depression of the S.T. segment, inversion of the T wave, and diminution of Q.R.S. amplitude. Changes occur in all four leads.

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INTRAPLEURAL FOREIGN BODIES*

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FOREIGN bodies in the bronchi and lungs are rather common but in the pleural cavity are comparatively rare.

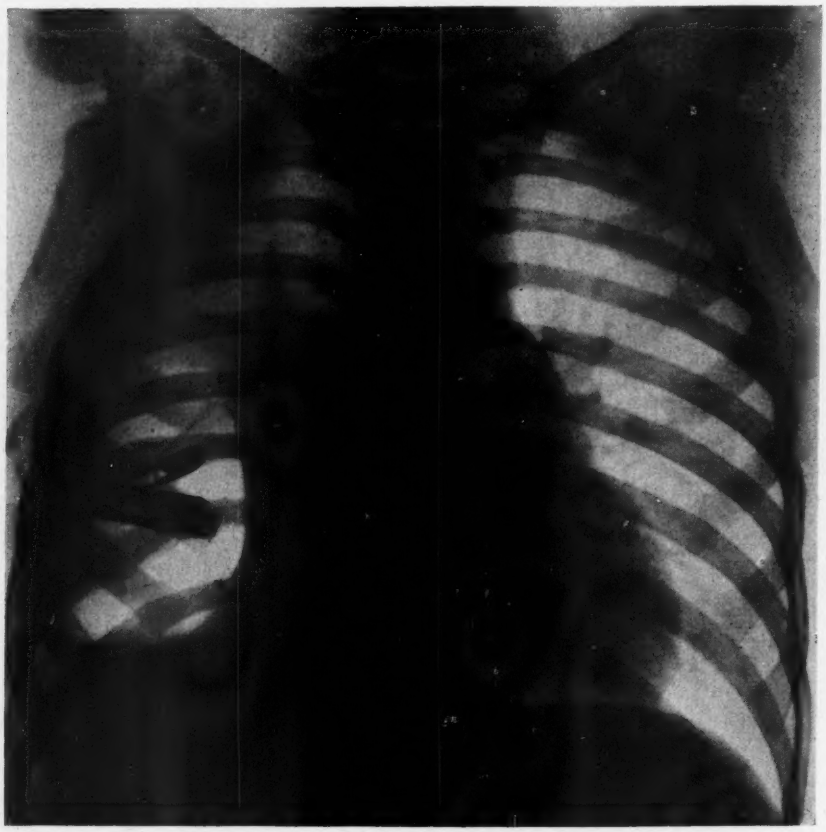

FIG. 1. Showing the empyema of the right chest with a rubber drainage tube extending into the empyema cavity. Just inferior to the drainage tube are two foreign bodies floating on the fluid surface.

The following case is of interest because of its difficult diagnosis and simplicity of treatment.

A boy, aged 4 years, entered the Children's Memorial Hospital November 16, 1937 with a diagnosis of early right lower lobe pneumonia. Twelve days after admittance the child developed a right sided empyema and was treated, on three occasions within the following nine days, by aspiration of pus. Cultures of the aspirated pus showed type 1 pneumococcus. On December 10, 1937 a right thoracotomy was performed under novocaine anesthesia and a rubber cylindrical tube inserted into the pleural cavity. The patient improved rapidly after this.

A roentgenogram of the chest taken January 17, 1938 (thirty-eight days after the thoracotomy), revealed three foreign bodies in the right pleural cavity at the costophrenic sinus. (Fig. 1.) In some of the roentgenograms taken later the bodies floated on top of the intrapleural fluid while in others they seemed to be at the bottom of the fluid. There were numerous suggestions as to what these foreign bodies

* From the Surgical Service of Dr. A. H. Montgomery at the Children's Memorial Hospital. Read before the Chicago Surgical Society, December 9, 1938.

were. It was thought at first that they might be pieces of a drainage tube which had become old and broken. However, an investigation in the operating room where the tube was inserted and in the ward where it might have been removed and replaced, revealed the fact that only new rubber tubing was used in this case. Also upon testing a rubber drainage tube in water it was found that the tube floated on top of the water and as the foreign bodies found in this case were sometimes seen at the bottom of the fluid in the films it was hardly possible that they were due to retained bits of tubing. No history was given of the presence of any foreign body in the upper respiratory passages which might have penetrated the lung and visceral pleura.

It was decided to remove the foreign bodies regardless of their composition. The original thoracotomy opening was still present and it was believed that the bodies could perhaps be most easily removed through it, possibly by an endoscope of the type used in bronchoscopic procedures. However, since the bodies seemed to float in some of the roentgenograms it was finally decided to attempt to wash them out by irrigation. The child was laid on his uninvolved side and normal saline irrigation through the thoracotomy wound was begun. A few seconds later the child gave a violent heaving cough and the irrigation fluid gushed from the wound carrying the foreign bodies out of the chest with it. They proved to be masses of zinc oxide ointment. Upon investigation it was found that this ointment had been applied to the skin around the drainage tube because of a dermatitis due to the pus escaping from the empyema. Roentgenograms taken after the irrigation showed the foreign bodies to be absent.

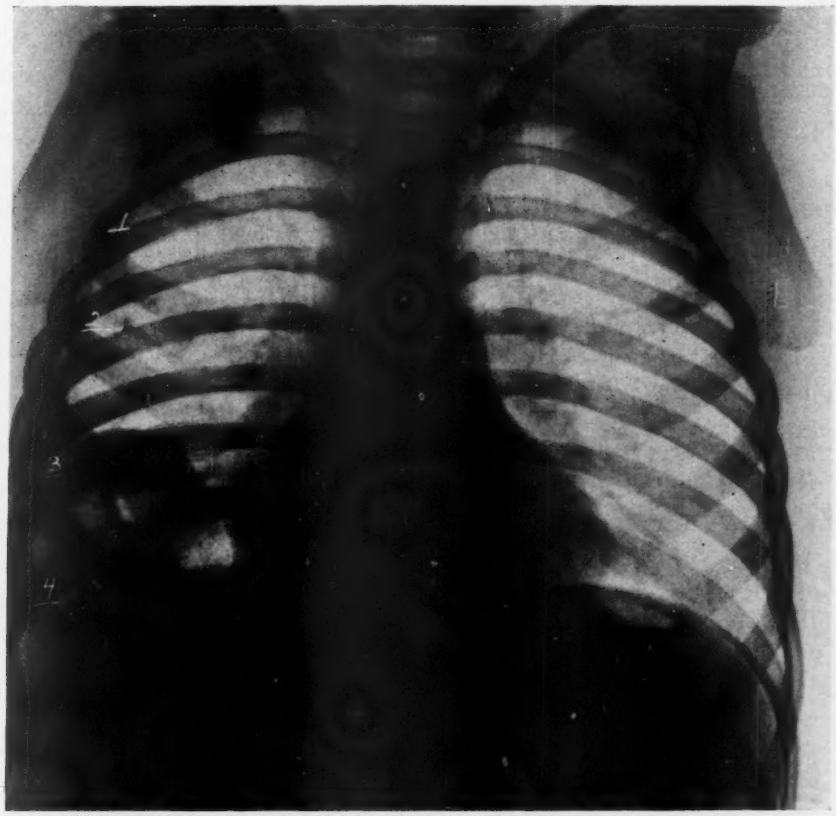

Fig. 2. The Arabic numerals 1, 2, 3 and 4 are opposite the oblong areas of zinc oxide ointment upon the skin of the posterior chest. These areas are 0.01, 0.25, 0.5 and 1 cm., respectively, in thickness.

The zinc oxide ointment was not considered before the irrigation as a cause of the opaque bodies. In order to show experimentally whether zinc ointment would cast a shadow in a roentgenogram layers of zinc oxide ointment of various thickness were placed upon the chest wall externally and roentgenograms taken through the chest. Figure 2 shows such a roentgenogram with zinc oxide ointment applied posteriorly upon the skin of the chest.

The Arabic numerals 1, 2, 3, and 4 are opposite the ointment oblongs which are 0.01, 0.25, 0.5, and 1 cm. respectively in thickness. The oblong of 0.01 cm. thickness is not visible, but the three areas of greater thicknesses are readily seen.

The child made an uneventful recovery and was discharged from the dispensary May 9, 1938.

Very few reports of intrapleural foreign bodies were found in the literature. They may be classified as (1) those entering the pleural cavity after having been inhaled, passing through the lung; and (2) those passing through the chest wall from the exterior.

Davidsohn,[1] in 1931, was able to collect reports of ten cases in the first group and added one of his own. These foreign bodies included straw, grass, rye, barley and other grain ears, and artificial teeth. His case was that of a deciduous tooth found in the pleura at necropsy in a woman aged 33 years. As a deciduous tooth is seldom shed after 12 or 13 years of age, the tooth in Davidsohn's case was probably in the pleural cavity for at least twenty years.

Graham[2] reported a case in which a child aged 18 months aspirated a branch of a tree. Part of the branch was removed at a thoracotomy operation but the remainder was found intrapleurally at necropsy.

In the second group of cases, that of foreign bodies penetrating through the chest wall from the exterior, are those due to accidental penetrations of the chest wall, missiles from fire arms, and unfortunate occurrences from surgical operation.

Andrews[3] had six needles break in doing 58,680 chest punctures. In one of his cases and in one reported by Passual,[4] an intrapleural broken needle was successfully removed by a thoracoscope. Rubber drainage tubes which have not been sufficiently anchored have not infrequently been found in the pleural cavity. Scheidel[5] removed a rubber drainage tube from the pleural cavity of a man aged 53 who had had the tube inserted seventeen years previously for empyema. The patient suffered much during the seventeen years from recurrent attacks of empyema. Jackson and Jackson[6] reported four cases of intrapleural rubber drainage tubes following operations on the chest. Pereira[7] successfully removed from the pleural cavity a wood splinter which entered as the result of a fall.

SUMMARY

Foreign bodies in the pleural cavity are comparatively rare.

A case herein reported is that of a child aged 4 years who suffered from pneumonia and a subsequent empyema. Thoracotomy and rubber tube drainage were instituted. Three foreign bodies within the pleural cavity were later seen in roentgenograms. These foreign bodies were floated out of the pleural cavity with normal saline solution through the thoracotomy wound and proved to be masses of zinc oxide ointment.

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PERINEURIAL FIBROBLASTOMA SITUATED AT THE JUGULAR FORAMEN*

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PRIMARY intracranial tumors that affect the nerves passing through the jugular foramen appear to be very rare, and a fairly exhaustive search of the literature reveals but few cases in which involvement of the tenth cranial nerve has been a part of the earliest clinical picture.

Stieda[1] in 1928 reported the case of a patient who first showed a weakness of the soft palate and a positive Romberg. The head was carried a bit to the right and the patient tended to fall to this side. This was followed in three months by bulbar speech and papilloedema. Five months later weakness of the face appeared. The patient was operated upon one year after the appearance of the first symptom with a fatal outcome. At post-mortem a tumor was found involving the dura of the right side of the posterior fossa and extending forward to the petrous ridge. The microscopic diagnosis was endothelioma.

Tschernyscheff and Rossels[2] reported, in 1931, a case in which the first symptom was hoarseness due to paralysis of the right vocal cord. This was followed in four years by a decrease in hearing on the right and headaches. Three years later atrophy of the right sternocleidomastoid muscle was noted. The patient died ten years after the onset of symptoms. Autopsy revealed a tremendous tumor involving the right clinoids, the lesser wing of the right sphenoid and the base to the foramen magnum. The microscopical diagnosis was osteosarcoma.

Cases have been reported by Seiffer and Koch[3] and Schwab[4] in which the last five cranial nerves were involved at about the same time. The former was a sarcoma and the latter a carcinoma. Schwabach and Bielchowsky[5] reported a case of involvement of the last eight cranial nerves by a myxofibroma.

A number of other authors have reported cases in which massive tumors have involved this region during their growth. Many of these have arisen from within the petrous pyramid or from outside the base of the skull. There is of course frequent involvement of this area by tumors of the acoustic nerve, that, increasing in size, grow downward, encroaching upon the region of the jugular foramen.

The case presented here is that of a perineurial fibroblastoma which originated at the site of the jugular foramen and involved the nerves passing through it some time before it encroached upon the nerves passing through the internal auditory meatus.

CASE REPORT

P. S., age 33, was admitted to the Neurosurgical Service August 17, 1927.

In January 1922, the patient had what he called a sore throat and became hoarse. He went to a physician and was told that one of his vocal cords was paralysed. His speech was greatly impaired at that time, but seemed to improve slightly during the next three months and then remained unchanged until admission. In 1924 he noted a marked impairment in hearing not accompanied by tinnitus or other local symptoms. In 1926, he noticed that his vision was not so good as it had been formerly and that he often saw double. At about this same time he became unsteady on his feet and frequently staggered, usually to the right. In September of that year he commenced to vomit, very often during one week, and thereafter about twice daily until the following January. The vomiting was not described as projectile. After January it became less frequent, until at the time of admission it occurred only once every four to six days. In

* From the Neurosurgical Service, University of Pennsylvania Hospital.

November 1926, he had occasional attacks of shaking of the head that lasted about five minutes and over which he had no control.

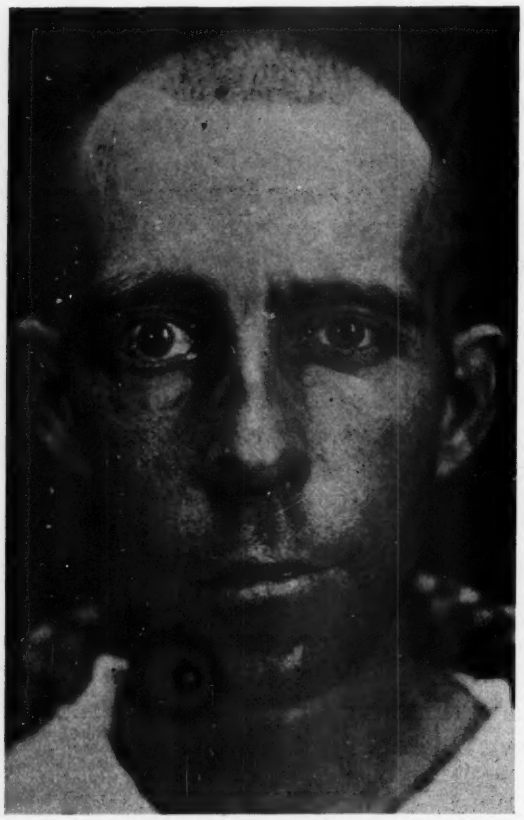


FIG. 1. Appearance of patient following operation.

These lasted up until the time of his admission. During the month prior to admission he had a numb feeling in both lips and tongue that was bilateral and slowly progressive. On admission he stated that he had felt increasingly weak and ambitionless during the previous year, and that he had experienced some loss of libido.

He was a painter by trade and had worked with lead paint for a number of years. His wife had had two miscarriages between two healthy children.

Blood pressure on admission was 142/90. Nothing of note was found in the general physical examination. Neurologic examination revealed a clear and alert mentality. Olfaction was normal on both sides. Visual acuity was $\frac{6}{9}$ in the left eye, and $\frac{6}{22}$ in the right. The visual fields showed slight concentric contractions. The right optic disc showed 8 diopters of choking and the left, 5 diopters. The veins of the retinae were engorged and a few small hemorrhages were present on both sides. The pupils were unequal in size, the right measuring 4.5 mm. and the left 4 mm. They reacted well to light and accommodation. There was horizontal nystagmus on lateral gaze to both sides, while on looking upward there were a few rotatory nystagmoid jerks. No limitations of the external ocular movements were noted, but there was a slight weakness of the right side of the face that included the eyebrow and forehead. The eighth nerve was entirely nonfunctioning on the right side. Taste was absent over the posterior third of the right side of the tongue. Sensation on the right side of the pharynx was much decreased, if not entirely lost, and the patient was unable to feel food when it lodged there. The palatal reflex was absent on the right, and the palate was drawn up and to the left on phonation. The voice was hoarse and monotonous. Dr. Gabriel Tucker reported the right vocal cord to be motionless and in the cadaveric position.

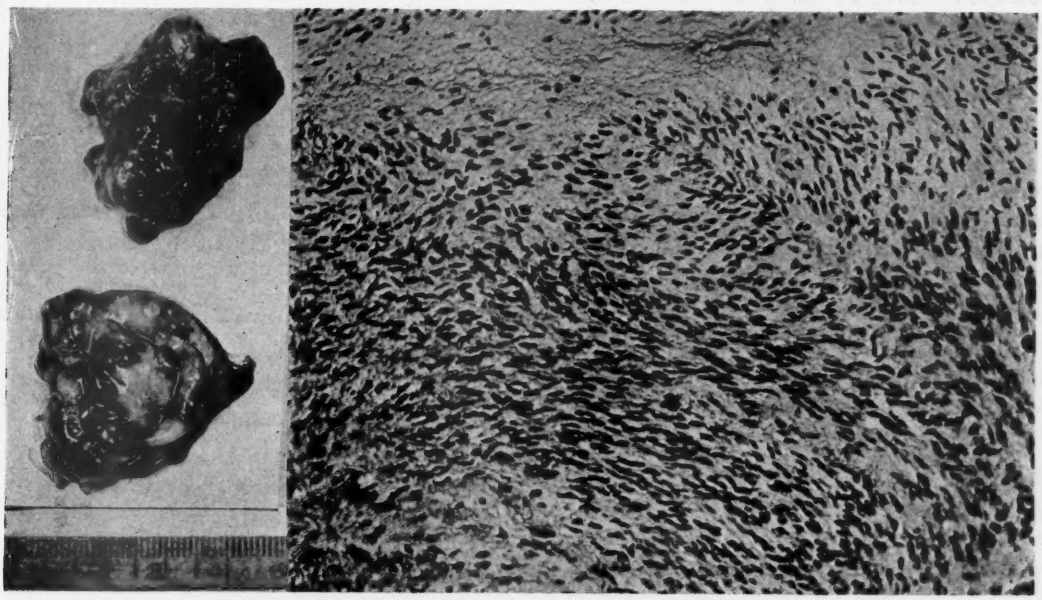
The muscles of the neck were strong on both sides and showed no atrophy, nor was any spasm noted. The tongue was protruded in the midline and showed no atrophy, fibrillation or tremor. Rapidly alternating movements were not done as well with the right arm as with the left, and the finger to nose test on the right side was a bit uncertain. In the vertical plane there was past pointing to the right with the right arm. The head was held inclined slightly to the right, but posture was otherwise normal. There was some swaying while standing on one foot, but there was no true Romberg's sign. When walking in a straight line, the patient staggered four times to the left and once to the right in eight attempts. No changes in sensation were found.

No biceps or triceps reflexes were elicited; the patellar reflex on the left was absent and on the right much decreased. The ankle jerks were equal and active. No pathologic reflexes were obtained.

Lumbar puncture in the lateral recumbent position showed an initial pressure of 260 mm. of water. Examination of the fluid revealed nothing abnormal. The white blood count was 6,600. The hemoglobin was 86 per cent. The urine showed a few hyaline casts.

On roentgen examination of the skull the posterior clinoids and the dorsum sellae were found atrophic. The pituitary fossa measured 12 × 16 mm. There were several areas of rarefaction in the posterior fossa, apparently above the nuchal ridge. There were some areas of calcification in the falx, and the calcified pineal body was not displaced from the midline.

A diagnosis of endothelioma of the cerebellopontine angle or neuroma of the tenth nerve was made and the patient was operated upon on August 24, 1927.



A B

FIG. 2. A, gross specimen. Note the glistening capsule. B, microscopic appearance of tumor, showing the tendency of the nuclei to arrange themselves in parallel rows. In the lower left corner is an area of degeneration, and in the upper right corner the cells are seen loosely packed without much ordered arrangement.

Operation. (Dr. F. C. Grant.) A bilateral suboccipital craniectomy was performed. The cross bow incision was carried well out beyond the tip of the right mastoid, and midline incision to the top of the third cervical spine.

The bone was rongeured away to the right as far as possible. The left ventricle was tapped and found to contain considerable fluid. The dura was opened and the occipital sinus tied. The rim of the foramen magnum and the arch of the atlas were then removed. Nearly all of the right cerebellar hemisphere was exposed and retracted gently, disclosing a hard, discrete, encapsulated, greenish-appearing tumor which had the right eleventh nerve running over its surface. We dissected the nerve off the tumor and could see the fibers of the ninth and tenth nerves running over the surface of the new growth. It was very easy to dissect the cerebellum back from the upper surface of the tumor, and we finally were able to isolate it and roll it somewhat laterally. We then made an incision in the capsule and curetted out the yellow, cheesy material which one often sees in tumors of this neighborhood. The tumor was freed up as much as possible and rolled away from the brain stem so that it could be lifted up quite readily with the pituitary rongeurs. We could not determine whether the seventh and eighth nerves ran through the tumor, for we could not see them at any time. However, it was quite obvious that the nerves running through the jugular foramen were involved. The tumor was removed from its cavity about this foramen with a blunt dissector, with an attempt to preserve the eleventh nerve. The removal of the tumor had no effect on the patient's respiration or pulse and his condition was extremely satisfactory. Hemostasis and closure completed the operation.

Following the operation there was complete paralysis of the seventh, eighth, ninth and tenth cranial nerves on the right. (Fig. 1.) The patient had some difficulty in swallowing and was fed with nasal tube for a few days, but convalescence was otherwise uncomplicated and he was discharged on September 18, 1927. At the time of discharge, in addition to the cranial nerve paralyses mentioned above, he had a suggestive cerebellar gait and a positive Romberg's sign. There was coarse horizontal nystagmus on lateral gaze, more marked on looking to the right. The optic discs showed a subsiding papilledema of 2 diopters. The other cranial

nerves were normal. Tendon reflexes were bilaterally diminished.

The patient was readmitted on December 29, 1927 for facial-hypoglossal nerve anastomosis. At that time he no longer had difficulty with his gait, and coördination was good. His condition was otherwise unchanged. Operation was performed on January 4, 1928, and he was discharged twelve days later.

A letter from him on January 10, 1937, states that he is well and working. His face in repose does not sag and he is able to take fluids without trouble. He has a small scar on his cornea that has been present for two years and has not increased in size. He has some difficulty in swallowing, his voice is still hoarse, he occasionally vomits and staggers in the dark, but these symptoms have not increased during the nine years.

Pathologic Report. The gross specimen was in four pieces, two of which are shown in Figure 2. The surface was nodular and tough, consisting of a whitish capsule 1.2 to 2.0 mm. in thickness. In places the capsule was ruptured. The cut surface showed a finely granular, yellow tissue very dense in some areas and soft and necrotic in others.

Microscopically, the tumor showed many areas of degeneration and contained many small cyst-like cavities. In places the tissue was moderately vascular, and some recent hemorrhages could be seen. Between the degenerated areas the growth was very cellular and the cells were arranged for the most part in parallel rows that streamed together. These rows were broken and separated in places by masses of similar cells loosely scattered with no ordered arrangement. (Fig. 2.) The nuclei of the closely packed cells were elongated, and there was a tendency for them to line up in rows, although no true pallisading was seen. The nuclei of the scattered cells were well rounded. In the protoplasm of the cells, fine collagen fibrils ran parallel to the long axes of the cells. Diagnosis: Perineurial fibroblastoma.

COMMENT

It is impossible to say from which nerve sheath this tumor arose, but the operative findings would limit it to those nerves passing out through the jugular foramen, and of these the eleventh was little, if at all, affected. In this connection the history of attacks of shaking of the head is interesting, as well as the fact that the patient held his head inclined slightly to the right. These signs might well be considered as due to irritation of the eleventh nerve caused by pressure from the tumor. It seems justified to assume that the nerve sheath from which the tumor arose was that of ninth or the tenth nerve.

The diagnosis of this type of tumor should not, in the majority of cases, offer any great difficulty, as the other causes of such a syndrome are few, and can for the most part be easily ruled out. If signs of increased intracranial pressure have not become apparent, the most likely condition producing a similar picture is inflammation of the common arachnoidal sheath of these nerves (Vernet, 1922[6]), or adenopathy of the glands lying around the jugular vein at its exit from the foramen. Both of these conditions are generally syphilitic and as such offer little difficulty in diagnosis if a Wassermann reaction is done. In cases of trauma, in the form of stab wounds or gunshot wounds of the back of the neck, the cause is obvious and the twelfth nerve is nearly always involved. Extracranial tumors of the nerves or of their sheaths, or of other adjacent structures in the neck may give a nearly identical clinical picture before signs of pressure appear. In some cases, in which the tumor in the neck is small and deep seated, a differentiation may not be possible before signs of pressure become apparent. Large tumors can usually be palpated without much difficulty if care is taken to relax the sternocleidomastoid muscle. These extracranial tumors will frequently be painful on palpation.

Tumors of the base of the skull are the most frequent cause of the syndrome when increased intracranial pressure is present, and if roentgenograms of the base of the skull show no changes in the bone a differential diagnosis is hardly possible. This is not, however, a tremendous disadvantage, as the operative approach in either case would be the same. It is important that the roentgenograms in these cases be taken in the Hirtz position, the so-called "base

plate," as the usual lateral views or those taken in the anteroposterior axis will fail to show any evidence of the slight bone changes seen in early cases. Abscess of the region of the cerebellopontine angle must be thought of, but the history together with the pulse and temperature will usually give the clue. Cysts and cysticercus have been mentioned in the literature[2] as being other possible causes of the syndrome of the jugular foramen.

The fact that this type of tumor is amenable to surgical intervention and can be removed entirely makes the early diagnosis a matter of paramount importance.

SUMMARY

A case of perineurial fibroblastoma lying at the inner end of the jugular foramen is reported. It probably arose from the sheath of the ninth and tenth nerves. The tumor was entirely removed and the patient is well and working after eleven years.

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PSEUDOPODIAL DIVERTICULA OF THE URINARY BLADDER*

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THE name pseudopodial diverticula alludes to the podial or false, foot-like projections of the diverticula originating from the bladder.

A careful search of the literature for cases of diverticula of this type produced no results, although herniae with diverticula were reported. Rongier[1] of Marion's clinic, Paris, reported a case of inguinal hernia with an infected diverticulum of the bladder containing a calculus. Polkey[2] discusses vesical diverticula and makes the positive statement that these may invade the inguinal or crural canals, but presents no cases. Samuels[3] presented a case of diverticula of the bladder herniating through the left inguinal ring.

Theories of etiology range from the view that congenital origin accounts for the condition to the opinion that obstruction and infection are the active causes.

Joly,[4] Watson,[5] Hyman,[6] Ward,[7] Randall,[8] Pugh,[9] and Moore[10] support the theory that diverticula are of congenital origin. Hinman[11] states that the potential breach in the wall of the bladder is present at birth and the mild distention with each normal micturition may widen it, without presupposing obstruction and increased intracystic pressure. Accessory ureteral buds, vestigial Wolffian ducts and patent urachi explain satisfactorily only a minority of diverticula.

Day and Martin,[12] Herbst, Polkey and Weller,[13] Boardman,[14] Walters and Mulholland,[15] Kutzmann,[16] and Rolnick and McNulty[17] subscribe to the acquired theory of the formation of vesical diverticula. Intrauterine defects of the bladder and obstruction may play important parts in vesical diverticulation. Herbst, Polkey and Weller produced experimentally diverticula of the urinary bladder in dogs and reached the conclusions that a moderate degree of obstruction, traumatic weakening of the bladder wall and increased intracystic pressure during micturition, such as may be produced by spasmodic contraction of the detrusor muscle due to infection, or other causes of increased muscle irritability, acting over a period of time are three important factors which evidently play important rôles in the formation of diverticula.

Close[18] adds another developmental element in diverticulosis to the five already known: temporary agglutination of urethral mucosa producing retention, superabundance of embryonic tissue (Fig. 1) vestigial Wolffian ducts, supernumerary ureteric buds and patent urachus causing traction from without the bladder. He states, "one must understand the nature of the endopelvic fascia to visualize the importance of the allantoic sheath of Delbet, that condensation of mesoderm which surrounds the bladder apex and laterally from the lateral margins of the empty bladder towards the obliterated umbilical artery. The development of this lateral sheet of fascia has occurred by the divergence in the embryo of the umbilical arteries from the allantois carrying away with them a process of the allantoic sheath from the whole of the allantois. In the filling adult bladder the slack of this process is gradually taken up by its expanding walls, when the whole allantoic sheath is utilized in the development of the bladder, the obliterated umbilical arteries lying in direct contact with the viscus. *Projection of this sheath* into the obturator foramen was found frequently in this *dissection*. Delbet

* Presented before the Section of Genitourinary Surgery, New York Academy of Medicine, December 21, 1938.

had mentioned it once. Three of five anatomical dissections of the pelves showed a considerable diverticulum bound to the hour-glass constriction in its midportion. There were five diverticula arising from the upper and four from the lower half of the bladder.

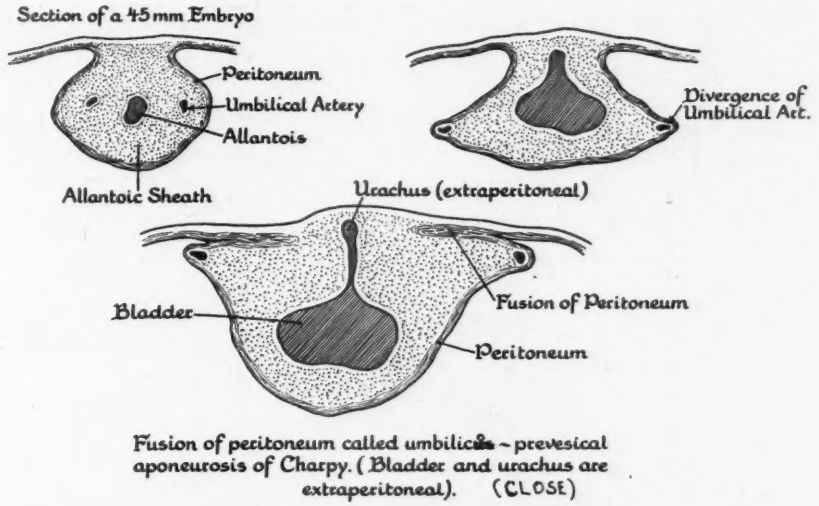

Fig. 1.

attached side, the apex of the mucosal protrusion being held firmly by such an attachment (endofascial). He concludes that traction of fibrous tissue, notably a developmental attachment of the allantoic sheath to the obturator foramen appears to have a predisposing influence in the formation of diverticulum. It was confirmed at operation and autopsy."

CASE REPORT

P. M., male, aged 64 years, was admitted to the hospital because of a painful swelling on the inner aspect of the right thigh which had been present for two days. This swelling was about the size of an orange, red, tender, fluctuating and fixed to the muscle tissue though not attached to the femur. Temperature was elevated, pulse rapid. A diagnosis of local abscess was made, the mass incised, and a large quantity of foul smelling purulent material evacuated. The cavity was drained.

Following this operation, the patient never voided. All of the urine drained through the wound, located on the inner aspect of the right thigh, 6 inches below Poupart's ligament. Cystoscopic study was contraindicated due to the septic temperature and debilitated general condition of the patient. A cystographic study, made after the injection of a 5 per cent solution of sodium iodide into the bladder, showed the bladder contracted in size and with an (Fig. 2.) Four of those arising from the upper half were of small size and resembled the ordinary type of diverticula. Number five was a large pyriform-shaped diverticulum arising from the right side of the upper half of the urinary bladder and extending down into the femoral region through the obturator foramen for 3 inches. Three of the four arising from the lower half of the bladder extended down into the right femoral region and the mid-thigh. The fourth (number nine) extended down into the left femoral region and was divided into three parts by two constrictions. These diverticula ranged in shape from pyriform to curvilinear sausage to pseudopodial forms and varied in size from 2 to 6 inches.

Suprapubic cystotomy was performed on May 19, 1932. A middle vertical suprapubic incision was made. Due to the inability to distend the bladder and to the firm fibrotic adhesions in the space of Retzius, the peritoneum was opened accidentally. This opening was utilized in locating the urinary bladder which was bound down behind the symphysis pubis. The peritoneum was then closed. The bladder was opened and a mushroom catheter inserted for drainage. The patient continued to run a septic temperature, ranging between 100 and 103.6 degrees. There was a large, tense, smooth, fluctuating swelling of the scrotum with a gangrenous area near the perineal attachment. There was also a brawny edematous induration of the perineum as well as of both groins.

With the patient in lithotomy position, two vertical incisions were made into both sides of the scrotum, each draining considerable pus.

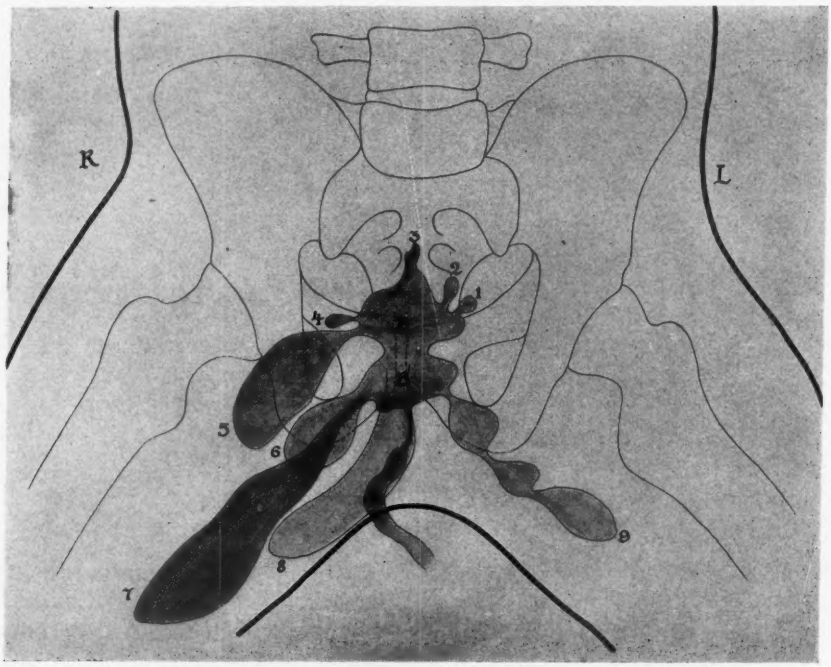

Fig. 2. Explanatory notes of urethrocystogram: *R*, right side; *L*, left side; *B*, bladder, hour-glass in shape; *U*, urethra; 1–9, diverticula of the bladder; 1–4, diverticula arose from the upper half of the bladder, were small in size and resembled the ordinary type of diverticula; 5, a large pyriform-shaped diverticulum arising from the right side of the upper half of the urinary bladder and extending down into the femoral region through the obturator foramen for 3 inches; 6–8 arising from the lower half of the bladder extended down into the right femoral region and the mid-thigh; 9, extended down into the left femoral region and divided into three parts by two constrictions.

Blunt dissection with the finger through these incisions showed that these cavities extended toward the inguinal region. A 1 inch vertical perineal incision to but not into the urethra, evacuated considerable pus. Further blunt separation through these incisions showed that the cavity communicated with the abscess cavities above. A No. 29 F. urethral sound was passed into the bladder and no stricture was noted. A mushroom catheter was inserted into the bladder and sutured to both the bladder wall and the abdominal wall.

The patient succumbed a few days later. An autopsy could not be obtained.

In the past thirty-two years much has been written about vesical diverticula, but in no instance has a case of multiple hernial formation of diverticula of the urinary bladder been reported.

Obstruction at the neck of the bladder, acting over a period of time, causing hypertrophy of the musculature of the bladder wall with cellulation and sacculation, and increased intracystic pressure produced by spasmodic contraction of the detrusor muscle due to infection, played important rôles in causing these divertuicla.

It is possible that the endofascial allantoic sheath of Delbet as described by Close acted as a guide for the diverticulum extending into the right obturator foramen. Following the path of least resistance between already depleted tissue elasticity, and compression and decompression by the muscular tissue on either side, the other diverticula-like moles burrowed snugly and quiescently into their respective positions.

The diverticula here reported were, in our opinion, of congenital origin.

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A roentgenographic unit should be a part of the well-planned convalescent hospital, and should be available constantly, for emergencies as well as routine work. Radiotherapy usually would be unnecessary.
From—"Convalescent Care" (New York Academy of Medicine).

URINARY LITHIASIS IN INFANCY AND EARLY CHILDHOOD*

WITH REPORT OF A CASE OCCURRING IN AN INFANT OF TWELVE MONTHS

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A CAREFUL study of the urinary tract in any large group of children will reveal an astonishing number with diseases requiring surgical treatment. Such disturbances are common and include practically all of the conditions occurring in adults. Much important work has been done in this field, particularly during the last few years. Considering the improved methods that have been developed for the diagnosis and treatment of these cases, and particularly the highly refined instruments that are now available, one is impressed with the thought that urology in children has become a specialty within a specialty.

In the civilized portions of the world, calculi in children are encountered infrequently at the present time. This statement applies particularly to North America. Calculi were not uncommon in England during the nineteenth century when stones were thought to be a disease of the extremes of life, being common among the very young and the very old. In Asia and certain parts of south eastern Europe, calculi are still prevalent. No doubt stones occur in America more frequently than the number of published cases would seem to indicate. It is probable that the syndrome of ureteral or renal colic in children may be mistaken for enteric colic. Bugbee and Wollstein[7] reviewed over 4000 necropsies on infants, finding calculi in the kidneys of thirteen, one of whom was only eleven days old. Clinically, Campbell found eight cases of calculi in a group of 580 children presenting urologic problems. For these observations it is apparent that careful urologic studies in infants and children will reveal the presence of calculi in a fairly substantial percentage of cases.

It is known that calculi may form during the period of gestation. Lagenbeck[13] reported a vesical calculus in a fetus of six months. Rayer[12] stated that renal calculi have been observed at all ages including the fetus and infant. He cited ten cases, two fetuses of six and eight months, three newborn infants ranging from one to eight days, five infants from three to twelve months of age. Bokay[14] has pointed out that during the first few days of the normal infant's life, the urine is extremely rich in uric acid and it is not until the end of the second week that the more usual characteristics of the urine are assumed. It is thought by some that the uric acid crystals or deposits may become nuclei for the formation of larger stones.

Most of the cases of calculi in children reported since 1900 have appeared in the older age groups. Thomas and Tanner[13] collected 203 cases of urinary calculi in children under 15 years of age. Their paper was the result of a general questionnaire sent to members of the American Urologic, American Pediatric and the Central States Pediatric Societies. The average age was found to be 7.8 years. Forty of the total were in children up to 5 years of age; sixty in children between 5 and 10, and fifty-eight in those between 10 and 15. The ages of forty-five were not given. Although forty cases occurred up to 5 years, no definite statements as to the ages of the children and the disposition of the stones are lacking. Campbell[3] has reported on a series of children up to 12 years of age, but does not state the ages of the children on whom he operated.

Several interesting examples of calculus disease occurring in the very young are

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recorded in the literature. André[19] reported the case of a boy 13 months old with a right ureteral calculus which required ureterolithotomy. Brown[20] recorded the case of a boy 3 years of age who had a stone in the lower portion of the left ureter. A shadow completely filling the pelvis of the right kidney, and a large sausage-shaped shadow in the lower portion of the left ureter were found on roentgenographic examination. A left ureterolithotomy was done and the sausage-shaped mass was found to consist of nine faceted stones. The convalescence was uneventful.

Hinman[21] reported the case of a girl eleven months old with renal and ureteral stones. The baby, born of healthy Austrian parents, after a normal pregnancy and labor, was breast fed for three months and then given diluted condensed milk. No water other than that contained in the milk was given. Suddenly at eight months, the child had complete retention of urine for three days accompanied by great suffering. The symptoms subsided quickly with the passage of a large quantity of urine and a round stone the size of a pea. The following day severe convulsions were followed by the passage of a second smaller stone, and later three other stones were spontaneously voided. The catheterized urine was cloudy. Microscopically it contained a few red blood cells and many leucocytes and rod-shaped organisms. A roentgenogram showed a shadow in the region of the left kidney and two others in the line of the lower portion of the left ureter. Cystoscopy proved these shadows to be calculi and the left kidney and ureter were removed.

REPORT OF A CASE

From a survey of the literature, it appears that the following case is one of the youngest, if not the youngest, case in which a ureteral calculus was found to be producing such a degree of obstruction as to demand urgent surgical removal.

A male infant, N. L., aged twelve months, was admitted to St. Mary's Hospital, Brooklyn, on December 15, 1937. The history obtained from his Mother showed that he had for the past seven days been irritable and lost appetite.

Fig. 1. Plain *x*-ray, showing large shadow of ureteral calculus lying in the right lower ureteral area.

Toward the end of the week, he seemed to have a high temperature accompanied by severe pain in the abdomen. He was seen, on the seventh day, by a pediatrician who found the temperature to be 105°F. On deep palpation of the abdomen an indistinct mass could be felt on the right side. Many pus cells and a few red blood cells were found in the urine. No casts or sugar were present. The child apparently had no difficulty in voiding.

Following his arrival at the hospital, an emergency urologic consultation was requested. When first seen, the child presented the picture described above and was critically ill. His pulse ranged between 150 and 160, and his temperature was 104.6°F. He was restless, irritable, appeared to be very toxic and dehydrated, and was in severe pain. The blood count was as follows: leucocytes 37,500, erythrocytes, 4,830,000, neutrophiles 49 per cent, eosinophiles 1 per cent, lymphocytes 49 per cent, monocytes 1 per cent. *Bacillus subtilus* and a few Gram-positive micrococci and diplococci were recovered on culture of the urine.

A plain roentgenogram and an intravenous urogram revealed an elongated shadow in the lower third of the right ureter. A diagnosis of a

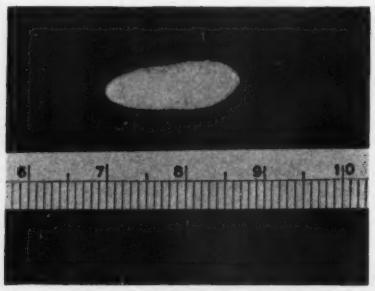

Fig. 2. Photograph of calculus.

Fig. 3. Child six months after operation.

calculus in the right lower ureter was made. (Fig. 1.) In view of the critical condition of the patient, the question arose as to whether the ureterolithotomy should be preceded by a nephrostomy. There was obviously serious urinary obstruction with infection in the renal pelvis and early drainage was clearly indicated. After due consideration, it was decided that removal of the stone might provide adequate drainage, particularly if the incision in the ureter were left wide open. This procedure proved to be a practical solution in this instance. The operation was performed within forty-eight hours of admission.

Under ether anesthesia, a right groin incision was made. The peritoneum was reflected to the midline. On palpation, the calculus was readily identified and milked back for a distance of approximately 4 cm., to make it more accessible for removal. A longitudinal incision, about 1 cm. in length, was made in the wall of the ureter. The calculus was removed without difficulty. (Fig. 2.) The incision in the ureter was not closed by suture because free drainage of the distended renal pelvis and ureter was desired and a Penrose drain was inserted. The wound was closed in layers.

Following the operation, the temperature dropped to 100 degrees on the second postoperative day. It was normal on the fourth day and remained so until he was discharged. Convalescence was essentially uneventful. The infant was dismissed on the eighteenth postoperative day. A chemical analysis of the calculus showed it to be composed mainly of calcium carbonate. The patient was seen six months later when he was in excellent health. (Fig. 3.) No pus or blood was found on examination of the urine and on palpation of the abdomen, no evidence of the original mass on the right side could be felt.

DISCUSSION

Etiologic Factors in Stone Formation. Stones vary not only in their chemical consistency, but in their size, shape, and position as well. There are many etiologic factors involved in the formation of the varying types. Among those theories given credence in recent literature are the following: (1) The lack of certain vitamin assimilation or utilization. In 1934, a series of experiments was carried out at the Cleveland Clinic in which rats were maintained on a diet deficient in vitamin A for 250 days. Post-mortem study revealed the presence of vesical calculi in 88 per cent and of renal calculi in 42 per cent of the experimental animals.[22] (2) The reduction of content of urinary colloids to the point where they are unable to maintain the urinary salts in solution. (3) The effect of faulty drainage and urinary stasis. (4) A disturbance of the calcium metabolism

brought about by dysfunction of the parathyroid gland. (5) Infection. Infection thrives in the presence of obstruction so that these two phenomena are usually associated. Any of the bacteria commonly found in the urinary tract are seen under these conditions. Probably the most prolific from the standpoint of stone formation is the *Bacillus proteus ammoniae*. This organism has the faculty of splitting the urea of the urine and liberating ammonia, thus making the urine intensely alkaline. Calcareous incrustations and stones composed chiefly of calcium phosphate are the result. (6) Randall's[5] theory is that calculogenesis is but a symptom of some form of renal papillary damage antedating the disposition of crystalline urinary salts. Microscopic studies have shown a hitherto unrecognized papillary lesion consisting of the deposition of calcium in the walls and intertubular spaces of the renal papilla. Once this calcium deposit has lost its epithelial covering it is subject to a constant bath of calyceal urine from which it is naturally assumed that crystals are deposited as upon any foreign body. The concretion grows and then breaks away from its point of origin and passes down the ureter.

Anomalies of the urinary tract undoubtedly predispose to calculus formation. Clinically Hyman[8] found congenital anomalies to be the underlying factor in an analysis of 150 instances of urologic maladies in children. A marked pyuria was associated with all the cases, demonstrating the fact that anomalous urinary organs are very prone to infection. The greatest number of lesions was found in the upper urinary tract. Joly[6] points out that ureteral calculi found in children are often associated with congenital strictures or other anomalies of the ureter. An interesting illustration of an anomaly associated with calculus formation is reported by Burstein.[2] An eighteen month old female had a double kidney with a Y shaped ureter. A calculus was found lodged at the juncture of the two ureters. A nephrectomy and ureterectomy resulted in recovery.

Chemically, almost all types of calculi have been found, among them being those composed of calcium oxalate, carbonates, phosphates, uric acid and cystine. The uric acid stone is common in the newborn and the cystine stone occurs more commonly before adolescence than afterward. Both types are very smooth and often pass easily through the ureter without becoming impacted. From a study of twenty-one cases of calculi in children, Kretschmer[9] found cystine calculi, pure or mixed, in five cases. Kretschmer agrees with Hyman[8] that cystinuria is a familial and hereditary disturbance of the mechanism of protein metabolism which is often observed in children and predisposed to the formation of cystine calculi. Joly[6] points out that it is most probable that the uric acid infarcts found in the kidneys of infants are not precursors of the type of calculi met in the kidney of the adult. The only type of calculus which he believes is definitely caused by a diathesis, is the cystine calculus.

As has been previously mentioned, calculi are usually associated with infection, under these circumstances stones are likely to be phosphatic. When the stone has not been formed in the presence of infection, it is generally composed of calcium oxalate. The calculus may have a little of each of these elements, but if it is mainly calcium oxalate it has almost surely been formed in an aseptic area. If it is composed in part of phosphates, then it has probably originated in an infected area. The amount of the phosphatic deposit indicates the length of time it has been lodged in the presence of infection. Most of the large stones are of the phosphatic type and grow more rapidly than other varieties.[6] The stones which Kretschmer[9] found in the ureter were composed of cystine or calcium oxalate or calcium oxalate with some phosphate. In one instance of a bladder calculus he found a trace of calcium

carbonate along with magnesium ammonium phosphate and calcium oxalate.

Urinary calculi in children, generally speaking, are not uncommon, but they are seldom found in the ureter. The calculus can usually pass easily through a child's ureter because of its ready dilatability. It is only the very large stones or those which become impacted, that must be removed surgically. Only 10 per cent of the stones formed in the kidney remain there. Of the 90 per cent that leave the kidney, only a few lodge in the ureter; therefore, the remainder of the calculi either are passed from the meatus, stop in the urethra, or remain in the bladder.[3] In summary, Martz and Lewis[16] state that 85 per cent of all kidney stones will pass spontaneously.

Most symptoms of calculus disease are observed during the migratory phases. In moving from the kidney the calculus causes obstruction and mechanical irritation. The back pressure from accumulated urine will cause a marked distention of the organs above the site of obstruction. If persistent, this may result in an enlarged ureter and destruction of renal parenchyma. Primary stones do not originate in the ureter, but in all probability secondary stones do. In children, where a definite urinary obstruction already exists, calculi above that site are usually multiple. Kretschmer also found that in this series five children, or 22.8 per cent, had stones in more than one organ. Both in children and adults calculi are found more commonly in the male than in the female. Jeanbrau[11] has found that the ratio in adults is 3 to 2. Thomas and Tanner[13] and Kretschmer[9] have reported a ratio of 3 to 1.

Diagnosis. The diagnosis of ureteral calculus in children may be difficult. It is frequently impossible to get a satisfactory history because of the lack of observation on the part of the attendant, and because the infant himself is unable to describe his difficulties. The symptoms in an older child vary but little from those noted in adults. The symptoms of a ureteral calculus should be distinguished from those of enteral colic. In the case of stones there is an absence of diarrhea and the attack will be shorter in duration.

Pain is usually the most important subjective symptom. The typical renal colic type, originating in the costovertebral angle and radiating anteriorly down toward the bladder, is often noted. Since renal colic is usually accompanied by vomiting, this may direct attention to the intestinal tract. Calculi that are more or less stationary in the kidney, ureter or bladder may give rise only to a dull ache in the loin or lower part of the abdomen. Such pains rarely radiate. With the migration of the stone, a smooth muscle spasm or colic is usually induced. When the stone is renal or ureteral, the radiation of pain is most often toward the groin, pubis or isolateral genitalia. The pain of migratory vesical stones is frequently referred to the tip of the penis and in the young, often directs attention of the genitalia.[3]

Although pain is a dominant symptom, certain exceptions have been observed. Thompson and Exley[15] report a case of calculus in the ureter of a 7 year 10 months old female who did not have pain. Mertz and Lewis[16] believe that pyuria is the most common symptom of stone in children, with colicky pain next. Pyuria, urinary frequency, dysuria and pain are generally characteristic of calculi. In the event of a severe case of dysuria, a few drops of blood may appear at the end of urination. This symptom is particularly severe when a bladder calculus is present. Prolapse of the rectum may accompany stranguary. Chills and fever are the usual systemic reaction to an acute urinary infection, especially one produced by obstruction. Hematuria is absent in 50 per cent of the cases.

The urinary findings are the most constant and reliable of all. The presence of pus and blood cells in the urine should always lead the clinician to an investigation of the urinary tract. Red blood cells, pus cells, albumin, bacteria and crystals are valuable links in the chain of evidence in cases of obscure abdominal pain.[9] Crystals in the

urine are particularly helpful in diagnosing a cystine calculus. A culture of the urine should be made to determine the presence of infection and the type of offending organism.

A roentgenographic examination is part of the routine study. Pure uric acid stones are not opaque to the Roentgen rays and this may add considerable difficulty to the diagnostic problem. Kretschmer[9] reports that in twenty-one cases of calculi the roentgenographic findings were positive in all but one. A complete urologic survey should be carried out, not only to determine the presence or absence of calculus, but for the purpose of demonstrating any other pathology which might be present. The ureter, whether directly or indirectly involved, may show hypertrophy, dilatation, duplication, elongation or angulation and loop formation with adhesive bands stretching across segments.[1]

Intravenous urography is often a decided aid in diagnosing calculi. This examination may be readily carried out with a minimum of inconvenience to the patient and in many instances will give one the desired information. Where any doubt remains in the mind of the examiner, there should be no hesitancy in making a cystoscopic examination. Certain calculi that are not opaque to Roentgen rays will be picked up on ureteral catheterization or seen as filling defects in the pyelograms. Obstructive lesions at the bladder neck, if present, are easily demonstrated. Cystoscopy may not be necessary in every case but when employed a general anaesthesia is usually required. The instruments now available for cystoscopy are so highly developed that the bladder of even the smallest infant may be successfully examined.

Treatment. Surgical treatment depends on the site and size of the calculus. Evidence of urinary obstruction with infection merits serious consideration of its early removal. The surgical procedures employed are essentially the same as those performed on adults; nephrectomy, pyelotomy, ureterotomy, and cystotomy. Children stand surgery as well as, if not better than, adults, providing certain principles are observed, particularly meticulous control of hemorrhage and the prevention of acidosis. Hemorrhage should be carefully avoided by scrupulous care in effecting hemostasis at the time of operation. If hemorrhage has occurred, the blood loss must be made up by transfusion. The conservation of body heat, the liberal use of transfusions and glucose for beginning acidosis have rendered even the most formidable procedures relatively safe.[3] Wound infections in children are seldom seen and when they do occur recovery is rapid.

SUMMARY

1. The recent literature concerning calculus disease in children has been reviewed and a case of ureteral calculus, requiring urgent surgical removal, in an infant 12 months of age has been reported.

2. Stone formation is frequently associated with (a) infection; (b) developmental anomalies of the urinary tract; (c) obstructive lesions.

3. Abdominal pain is probably the most frequent subjective symptom. The presence of pus and blood cells in the urine is the most constant objective finding and indicates the need for further investigation.

4. The management and treatment of urinary calculi in children is essentially the same as in adults. Complete studies should be carried out to determine whether or not anomalies or obstructive lesions are present.

5. When properly handled, children tolerate surgical procedures as well as, if not better than, adults.

6. It would appear that ureteral stones in infancy are probably more common than is generally suspected. Those requiring surgical removal are rare.

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FEMORAL HERNIA IN INFANCY

CASE REPORT

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THE patient was a colored female infant born in the Harrisburg Hospital August 1, 1938, whose weight at birth was 4 pounds 3 ounces. She received premature care, developed normally and at the time of her discharge from the hospital on August 16, 1938, weighed 5 pounds and ¼ ounce.

She remained perfectly well until the night of October 8, 1938 when she began to cry as if in pain. The mother noticed a swelling in the left groin. When she touched the swelling the baby cried with more intensity.

Physical examination on admission revealed a swelling the size of a walnut in the left groin below Poupart's ligament. This swelling was firm and tender, did not transmit light, was dull to percussion and irreducible by taxis. The diagnosis of left strangulated femoral hernia was made.

Under general ether anesthesia a small incision was made parallel to and below Poupart's ligament. The diagnosis of femoral hernia was confirmed. The hernial sac was isolated and incised and found to contain the left Fallopian tube, the left ovary and a small parovarian cyst, the latter being somewhat discolored. The ectopic organs were replaced in the peritoneal cavity, the sac was ligated and the femoral ring was closed with two mattress sutures of 0 chromic catgut which were reenforced with a third suture. The superficial tissues were closed with interrupted fine catgut sutures and the skin with fine interrupted wire. A water-tight occlusive dressing was applied.

The child made an uneventful recovery. She was kept on breast milk with the addition of a complementary formula during her hospitalization. There was no postoperative vomiting or distention. The sutures were removed on the fourteenth postoperative day at which time the incision was healed completely, and the patient was discharged on the eighteenth day.

Incidence. Erdman[9] states that only 1 per cent of all femoral hernias occur in childhood. Watson[27] and Tasche[24] find them very rare before the twentieth year and Tasche did not believe that femoral hernias have been reported in any individual under 2 years of age. Denoeux[8] cites a paper by Werhner who collected 5,341 cases of femoral hernia, none of which were in children under 1 year of age, seven occurring between the ages of 1 and 5, and thirty-four between the ages of 6 and 10. Lloyd[16] discovered only one example of femoral hernia in three years at the Hospital for Sick Children, London; this was in a 5 year old male. Herzfeld[11] reported a series of 1,000 cases of hernia in children up to 12 years of age, only four of which were femoral hernias. Balch[1] discovered three cases of femoral hernia in patients under 10 years of age in a series of 100 cases of femoral hernia occurring at the Massachusetts General Hospital between 1924 and 1933. In Shawan and Altman's[23] series of ninety-seven cases, the youngest patient was 19 years of age. Coley[4] found 101 cases of femoral hernia in a series of 3,000 cases, twenty-one of which were in patients under 15 years of age. Rutherford[21] reported a series of 1,098 cases of hernia from the Belgrave Hospital, London, where the age limit for admission is 10 for girls and 12 for boys. He found only five cases of femoral hernia in the entire group. Rutherford also cited the statistics of Ribera y Sans from the Hospital l'Enfant Jesus, Madrid, who reported fourteen cases of femoral hernia out of a total of 3,098 cases of hernia. Horsley[12] states that although any type of hernia seen in the adult may be encountered in the practice of pediatrics, femoral hernias are seen very rarely. Approximately 70 per cent of all hernias seen in children are of the indirect inguinal type and 30 per cent of the umbilical type.

Indirect evidence of the rarity of femoral hernias in infancy is found in the thorough review articles[14,17,18,20] of strangulated hernias in infancy; in none of these articles is there a case of femoral hernia reported.

Sex. Cumston[6] says that after the age of 25 femoral hernias are more frequently seen in women than in men, but that in young subjects the defect is more frequently observed in boys than in girls. In Coley's series[4] eleven of the twenty-one cases occurring under 15 years of age were in boys and ten in girls. Four of Rutherford's[21] cases were females and the fifth was a male. Herzfeld[11] also found four cases in females and a fifth in a male. Watson[27] feels that femoral hernias in infancy are as common in one sex as in the other.

Side Involved. Femoral hernias usually occur on the right side, according to Erdman[9] twice as often as on the left. Watson[27] states that femoral hernias in children and infants are usually unilateral, but Coley[4] found two cases of bilateral involvement in male children.

Pathogenesis. There is a great deal of disagreement as to whether or not femoral hernias can be congenital in origin. Cowell,[5] Tasche,[24] and Denoeux[8] all feel that a congenital theory cannot be advanced as a cause. Tasche says that no femoral diverticulae are ever found in otherwise normal newborn infants. They find femoral hernias increasing in number with advancing years, in patients who have large increases in body weight with consequent loss of muscle tone, in patients, who suffer with chronic debilitating diseases, especially asthma. They also point out that these hernias occur far more often in parous women than in the childless.

On the other hand, Watson[27] feels that the congenital origin of femoral hernias is most widely accepted today. He is supported by Murray[19] who, in 200 consecutive necropsies, found fifty-two bodies with peritoneal femoral diverticulae, fourteen of these presenting the defect bilaterally. Watson feels that the predisposing factors are the presence of a preformed sac or prominence of a peritoneal depression due to lack of fat. He also cites absence of the lymph gland of Rosenmüller, a large saphenous opening or a large compressible femoral vein as possible predisposing factors.

A number of authors[3,24] have pointed to the regularity with which a pad of adipose tissue is found in association with a femoral sac. These men seem to feel that this lipomatous pad is in some way linked to the occurrence of the hernia.

Velasco Blanco and Eschegarez[26] think that femoral hernias in children may be either acquired or congenital. In the former there is an outward propulsion of peritoneum, while in the latter there is a preformed peritoneal diverticulum.

Diagnosis. The position of the hernial mass is very important in arriving at a diagnosis. Femoral hernias lie below Poupart's ligament and external to the pubic spine, whereas inguinal hernias lie above Poupart's ligament and internal to the pubic spine. With reducible hernias pressure upward and outward is required to accomplish reduction of an inguinal hernia, while pressure downward and backward is necessary to reduce a femoral hernia.

The differential diagnosis has been thoroughly discussed by various authors.[1,5,9,12,25,27] The chief considerations are inguinal hernia; psoas abscess where we often see spinal symptoms, gurgling in the mass being absent; lymphadenopathy in which we find no gurgling, no impulse with increased intra-abdominal pressure, no neck to the mass and frequently several nodes in a group; varix of the saphenous vein, in which there is filling from below; lipomas, dough-like in consistency and frequently bosselated; and a few other infrequent conditions such as undescended testicles, obturator hernias and cysts of the inguinal canal or of the canal of Nuck.

Contents. Omentum and small intestine are most commonly found, but in extremely young infants the omentum is not well developed. Rutherford's[21] case in a 4 year

old female contained only omentum. However, at some time or other almost any of the abdominal viscerae have been reported in femoral hernias.

Gore[10] and Watson[27] call attention to the fact that frequently the sacs of femoral hernias are empty. Lloyd[16] reported a case of strangulation of an empty sac in a 5 year old boy.

The Fallopian tube is not often found in the sac of a femoral hernia because in a normal anatomic arrangement the tube lies at a lower level than the femoral ring. Several interesting cases have been reported: Baty[2] found a well developed Fallopian tube strangulated in a femoral hernia in a male patient 29 years of age. Johnston[13] found the right tube incarcerated in a 30 year old female. Denoeux[8] cites a case report by Pillon who found a tube, ovary and appendix strangulated in a child 21 months of age. Daggett[7] found a tube and ovary incarcerated in a right inguinal hernia in a baby 7 weeks of age.

Treatment. All writers are agreed that the use of a truss has no place in the treatment of a femoral hernia in infancy and childhood. Operative treatment is the only successful method. Herzfeld[11] recommends exposure of the sac through the inguinal canal and makes no attempt to close the actual gap in the crural ring. She feels that ligation of the sac is a sufficient procedure. Coley[4] uses the femoral approach with closure of the femoral canal by a purse-string suture. He feels that this is the simplest procedure and his incidence of recurrence is no higher than that of others who use other techniques.

SUMMARY

1. Femoral hernias are of very rare occurrence in infancy, the patient reported here being probably one of the youngest in the literature.

2. The occurrence of a femoral hernia in an infant so young tends to lend weight to the theory that femoral hernia can be congenital in origin.

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STRUMA LYMPHOMATOSA (HASHIMOTO)*

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AMONG the most interesting diseases affecting the thyroid gland is that of chronic nonspecific thyroiditis. This condition went unrecognized as an entity until 1896, when Riedel[1] described a thyroid gland which was characterized by enlargement, iron hardness and extensive adhesions surrounding and adherent to it. When removing the gland, Riedel felt that he was dealing with a malignant neoplasm, but on histologic examination the pathology was interpreted as a chronic inflammatory process. The fact that this was benign and non-neoplastic was confirmed by the clinical course, since the thyroid had only been partially removed. Because of the marked firmness of the gland, Riedel referred to this condition as "eisenharte strumitis."

In 1912, under the title of "struma lymphomatosa," Hashimoto[2] described four cases having similar subjective symptoms to those reported by Riedel. These occurred in women all over 40 years of age. The disease was slow, insidious in onset and at operation was adherent to the trachea but not to the other surrounding structures. Following operation during which both lobes of the thyroid gland were removed in all four cases, there was a slow recovery which took twelve to eighteen months. Symptoms suggestive of postoperative hypothyroidism developed in two patients. Microscopic examination of the gland revealed a diffuse lymphocytic infiltration with numerous lymphoid follicles and germinal centers. Some degree of fibrosis, atrophy and hypertrophy of the glandular tissue was noted. Hashimoto found no specific etiology for the condition, but felt that this disease was a different entity from that of Riedel's struma.

Considerable argument has ensued concerning the possible relationship between these two conditions. In 1922, Ewing[3] suggested that struma lymphomatosa was an early stage in the development of Riedel's struma. Williamson and Pearse[4] and Jaffe[5] came to a similar conclusion. However, there have been opposite viewpoints especially by Joll[6] and Graham and McCullagh.[7] The latter authors point out that struma lymphomatosa is found almost entirely in women over 45 years of age, but that Riedel's struma, although usually found in adults, may occur at any age and is noted almost as frequently in men as in women. They state further that Hashimoto's struma is a diffuse one, whereas Riedel's struma may involve an entire or part of a lobe. These observers note also that in lymphadenoid goiter there is a diffuse formation of a delicate connective tissue in the later stages, whereas dense connective tissue may be found in the earlier stages of Riedel's struma. Finally these authors state that Hashimoto's struma operated or unoperated upon tends to develop signs and symptoms of hypothyroidism while Riedel's struma will rarely do so.

The etiology of both conditions is still unsettled. Riedel's struma appears to have some definite infectious factor. Graham[8] states that Riedel's struma "may be looked upon as having a local inflammatory process in the thyroid for which an etiological factor should be sought. The general body economy is affected secondarily by reason of destruction of the thyroid,

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interference with respiration and deglutition, and injuries to important blood vessels and nerves."

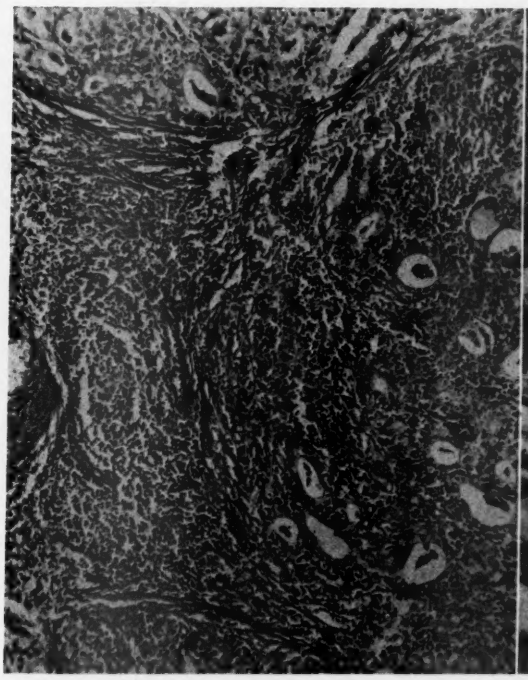

Fig. 1. View showing diffuse lymphocytic infiltration, some fibrosis and decreased number of thyroid acini.

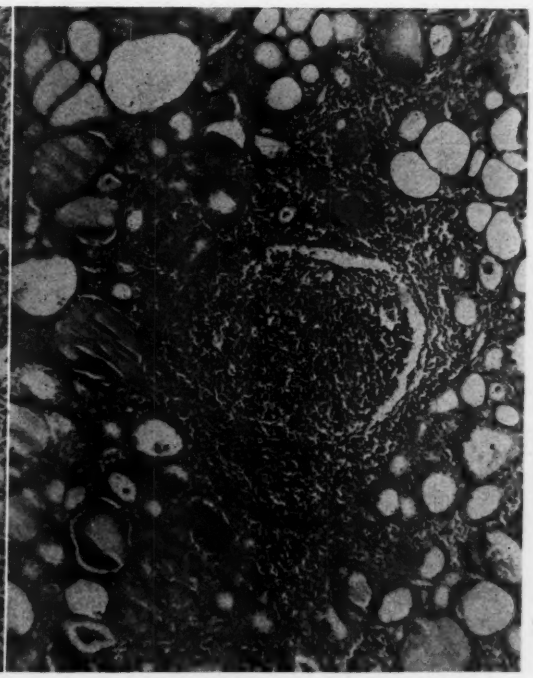

Fig. 2. Section showing lymphoid follicle with large germinal center in which there is active proliferation.

Williamson and Pearse[4] suggested that Hashimoto's struma is a pathologic variant of a lymphogenic secretion which is a normal function of the thyroid gland. McCarrison[9,10] was able to produce a condition simulating lymphadenoid goiter in rats by feeding them a diet deficient in vitamins but containing iodine. Graham and McCullagh[7] consider also that an antecedent hyperthyroidism may be a precursor to struma lymphomatosa and Graham[8] further states that "Hashimoto's type of lesion may be considered to be a local manifestation of a constitutional disorder—the changes in the thyroid tend to become degenerative rather than inflammatory and sclerosing." Jaffé[5] believes that any infection may lead to nonspecific degenerative changes of the thyroid gland with a secondary lymphatic reaction. He thinks further that those glands are predisposed to degeneration which are either hypoplastic or the site of a pathologic or physiologic involution. He tries to account for the disease's occurring mainly in women after the menopause by this predisposing involution.

CASE REPORT

L. R., a colored female, age 36, was admitted to the hospital on August 29, 1938 with a complaint of swelling in the neck for several years past. During the month before admission she experienced symptoms of choking and insomnia, and occasional attacks of palpitation and fatigability. Her past and family histories were irrelevant.

She was fairly well developed and did not appear acutely ill. There was no exophthalmos or other ocular sign of hyperthyroidism. The pupils were equal and reacted well to light and accommodation. The tonsils were somewhat infected. The thyroid gland was diffusely enlarged, lobulated and very firm. The lungs were clear, the heart not enlarged, but a systolic murmur was heard over the mitral area. Temperature was 98.6°F., pulse 85, respiration 18 and the blood pressure 190/125.

Urine examination was negative as was the blood Wassermann. The electrocardiogram showed some changes suggestive of myocardial disease. *X*-ray examination revealed no signs of pressure or displacement of the trachea, and

there was no evidence of intrathoracic goiter. The basal metabolic rate was plus 8.

On September 17, 1938, a total resection of the thyroid gland was done. The thyroid was enlarged and hard in consistency. It was not adherent to the surrounding structures except for some adhesions between the isthmus and the anterior surface of the trachea.

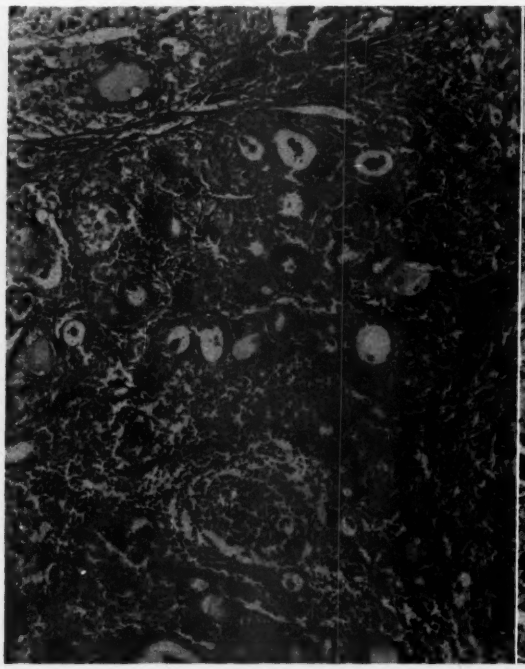

FIG. 3. Section of thyroid gland showing lymphoid follicle, lymphoid cell infiltration between acini and atrophy of acini with reduction of the colloid content.

FIG. 4. Groups of acini being infiltrated by lymphocytes, showing the reduced amount of colloid, which is often basophilic, and the peculiar large degenerating epithelial cells.

On the day following the operation the patient's temperature went up to 102°F. but dropped again very quickly. No other complications occurred. The wound healed well and she was discharged seven days following the operation. Since her discharge she has remained well and has shown no signs of hypothyroidism.

Pathologic Report. The specimen consisted of both lobes of the thyroid gland with the isthmus. It was half moon shaped and measured 13 × 4 × 2 cm. The gland was very firm in consistency and well encapsulated. The cut surface was rather pale pink and homogeneous throughout. No colloid material could be seen on gross examination. Microscopic examination showed a diffuse infiltration of the stroma with lymphocytes, which, however, varied in amount in different parts of the sections. In some situations the cells occurred in large numbers and between them atrophic acini were present. (Fig. 1.) In other microscopic fields the lymphocytes were less abundant, appearing as loose accumulations in the interstitial tissue, and the acini were not compressed. Here and there lymph nodules with germinal centers were noted. (Fig. 2.) The acini were in general uniformly small and contained either no colloid or reduced amounts. (Fig. 3.) The colloid stained poorly and in many instances was basophilic. There was some increase in the interlobular connective tissue stroma. The acini were normal in size, lined by flat epithelial cells and contained colloid. Where there was greater infiltration the acini were atrophic and lined by relatively large pale, acidophilic cells with granular cytoplasm, irregular nuclei and contained reduced amounts of colloid. (Fig. 4.) In some instances epithelial cells and lymphocytes were seen within the lumina.

COMMENT

Hashimoto's struma is almost always found in women. The average age in Graham's[8] and McClintock and Wright's[11] combined series of fifty cases was 49.2 years. They found that the symptoms were usually present for an average of sixteen months, with the duration of goiter slightly over four years. These authors

noted that in the fifty reported cases of struma lymphomatosa, all had involvement of both lobes of the thyroid and three-fourths of those suffering from Hashimoto's disease developed hypothyroidism following removal of the gland.

In contrast to these findings in Riedel's struma, the authors noted that sex was not an important factor and the average age of individuals suffering from this disease was 40 years. They found that in Riedel's struma the average duration of symptoms was about seven months while the goiter was present for an average period of 3.5 years. In this condition, the process, in approximately 30 per cent of cases was found to involve one lobe only and following operation about 26 per cent developed hypothyroidism.

McClintock and Wright[11] believe that there are certain signs and symptoms which may differentiate these two conditions, Riedel's struma is tender, very firm, fixed and resistant. Hashimoto's struma is resilient and more often gives symptoms of weakness and fatigue. Both types may cause dyspnea, dysphagia and a feeling of tightness about the neck. Patients with Riedel's struma following surgical treatment tend to improve rapidly and completely, whereas those with Hashimoto's disease have a long convalescence with frequent development of hypothyroidism.

In Hashimoto's disease, the shape of the gland is preserved. Both lobes and the isthmus of the thyroid are usually enlarged. The enlargement is intracapsular and the gland is not adherent to surrounding structures except occasionally to the trachea. The consistency is firm and the gland cuts with increased resistance. The tissue is pinkish white in color and definite trabeculae can be seen. Normal thyroid tissue cannot be made out grossly. Microscopically, although some fibrosis is present, the characteristic picture is that of a diffuse lymphoid infiltration between the acini. Numerous secondary lymphoid follicles are present and these may be very large in size. The lymphocytic infiltration varies in different parts of the gland. It is most marked in the interacinar tissues and in other parts of the gland it may be minimal in amount or totally absent. In the dense portions of lymphocytic infiltration, the acini are compressed and atrophic. The epithelial cells of these atrophic acini are peculiarly acidophilic, large, and have an increase in cytoplasm and a hyperchromatic nucleus. These cells are similar to those seen in degenerative processes. There is a diminution or absence of the amount of colloid within these acini. Only in occasional instances are plasma cells present. Organization of the lymphoid tissue into a definite node does not occur. In patients with hyperthyroidism, especially those who have taken considerable iodine, it is not uncommon to notice lymphocytic infiltration or even follicle formation. Differentiation can be made from struma lymphomatosa by the lack of thyroid hyperplasia, and atrophy of the thyroid tissue with the peculiar acidophilic cells described above in the latter condition.

Riedel's struma has a tendency to become fixed by adhesions to the neighboring structures but very seldom to the skin. The gland is very hard in consistency and because of this is often mistaken for malignancy although it does not have the irregular nodularity of cancer. The borders of the organ are not very definite and this finding is more marked when the gland becomes attached to the surrounding structures. The capsule is usually thickened. The cut surface is white in color and normal thyroid tissue is not evident grossly. On histologic examination, the characteristic picture is that of fibrosis. The fibrosis may vary from an increase in the interacinar and interlobular connective tissue to complete replacement of the thyroid parenchyma by dense connective tissue. The fibrous tissue is most marked beneath the capsule. There may be smaller or larger amounts of lymphocytic infiltration with occasional plasma cells. The acini which are still present are atrophic and show evidence of degeneration. Occasional areas of epithelial

hyperplasia may be noted. Giant cells frequently occur in the region of degenerating acini. Lee[12] believes that these are due to fusion of epithelial cells about colloid droplets.

SUMMARY

A case of struma lymphomatosa (Hashimoto) is reported with the clinical and pathologic findings.

This disease is uncommon.

The characteristic pathologic finding is a firm thyroid gland which microscopically shows a diffuse lymphocytic infiltration with lymphoid nodules.

Struma lymphomatosa is considered by some to be an early stage of Riedel's struma, but there are an increasing number of observers who feel that these conditions are separate and distinct diseases.

The etiology of Hashimoto's disease is as yet unknown. A general comparison of the clinical and pathologic differences between Hashimoto's and Riedel's struma is given.

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APLASTIC KIDNEYS*

CASE REPORT

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APLASIA of the kidney is always an interesting condition and at times one which presents a problem in diagnosis. In the present case the differential diagnosis had to be made between aplasia and tuberculosis of the kidney.

FIG. 1. Flat *x*-ray of the KUB tract. The right kidney is well visualized and slightly larger than normal in size. In the left kidney region an irregular, dumbbell-shaped mass is noted.

FIG. 2. Five minute excretory urogram shows good function and normal structure on the right side. No dye is visible on the left.

CASE REPORT

Mrs. G. M., 31, was admitted to the Brady Urological Service on October 11, 1938 complaining of pain in the *right* costovertebral region. Her illness had developed one month before admission with pain the only symptom present. It was dull, localized, and intermittent in character. Three weeks before admission, an intravenous pyelographic study done elsewhere had shown no excretion of dye from the *left* kidney.

At age 12, the patient had spent three years in a sanatorium for the treatment of pulmonary tuberculosis. Since that time she had been free from any clinical evidence of tuberculosis. Pulmonary *x*-rays taken eighteen months previously were reported negative.

Appendectomy and removal of an ovarian cyst were performed in 1936. At this time a bicornuate uterus was found.

On physical examination the lower pole of the right kidney was palpable and slightly tender to deep pressure. The left kidney was not palpable or tender. No masses were detected in the abdomen.

Laboratory Findings. In the bladder urine, a few white blood cells were found. Sedimentation of the bladder urine and stain for tubercle bacilli were negative on two occasions. Cultures of urine from the bladder and right ureter were

* Presented before the Section of Genitourinary Surgery, New York Academy of Medicine, November 16, 1938.

sterile. Vaginal secretion showed many white blood cells, but no gonococci and no trichomonas. A three hour P.S.P. test showed 68 per cent

FIG. 3. Cystogram shows a smooth defect on the left side of the bladder.

of dye eliminated, with a normal curve. Blood urea was 11 mg. per 100 c.c., blood sugar 67 mg. per 100 c.c. The hemoglobin was 94 per cent, red blood count 4,600,000, white blood count 6,200, with adult polys 62 per cent; immature 6 per cent; lymphocytes 30 per cent, monocytes 2 per cent. The Kline test was negative.

X-ray Examination. A chest film revealed no evidence of recent or active tuberculosis. The excretory pyelograms showed good and normal function from the right side, no function at any time from the left side, but multiple rounded calcified cyst-like shadows in the plane of the mid-abdomen. The bladder shadow was deformed on the left side.

At cystoscopy the bladder was normal. There was no evidence of inflammation or ulceration. No left ureteral orifice could be found. Indigo carmine, injected intravenously, failed to be excreted from the left side. Retrograde pyelography added nothing to the evidence obtained by excretory pyelography.

Seven days after admission 150 c.c. of air was injected into the left renal fossa and stereoscopic plates were made. This study failed to give much additional information. The cyst-like shadows previously described were of about the same appearance. The psoas shadows were clearly defined.

Exploratory operation was done eleven days after admission, by the lumbar route. Upon opening Gerota's fascia posteriorly, palpation in

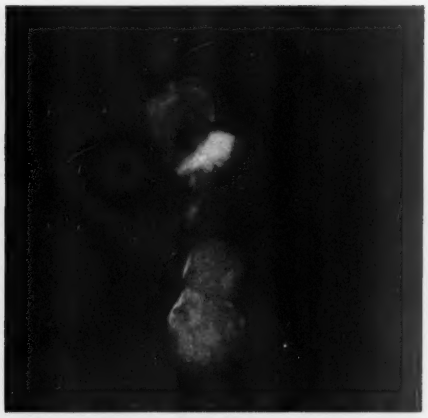

FIG. 4. *X*-ray of the tissue removed at operation.

the renal fossa revealed two small cyst-like masses. The superior mass was dissected free from the surrounding fatty tissue and was found to connect with a lower larger cyst-like mass by means of a thick band of fibrous tissue. The whole mass was dissected free and found to be attached to a structure having the appearance of an infantile ureter. This structure was ligated and cut, freeing the mass which was then removed in toto. Further exploration of this left renal fossa revealed no further evidence of a kidney.

The patient made an uneventful convalescence and was discharged as cured fourteen days after operation.

Pathologic Examination. In one of five sections, a small amount of adult kidney tissue was found. Throughout the other sections there were numerous small and large duct-like structures lined by columnar epithelium and surrounded by muscle. Numerous blood vessels were noted throughout the fibromuscular stroma. The cysts were lined by a layer of calcified tissue which in some fields had gone into bone formation. The tissue connecting the cysts was loose, infiltrated with lymphocytes, and there were several fields of hemorrhage.

SUMMARY

In favor of the diagnosis of a functionless tuberculous kidney on the left were: (1) the history of previous pulmonary tuberculosis; (2) absence of function from

the left kidney; (3) calcified areas in the left kidney region.

Against the diagnosis of tuberculosis were: (1) the absence of urinary symptoms; (2) the normal bladder mucosa; (3) failure to find tubercle organism in the urine.

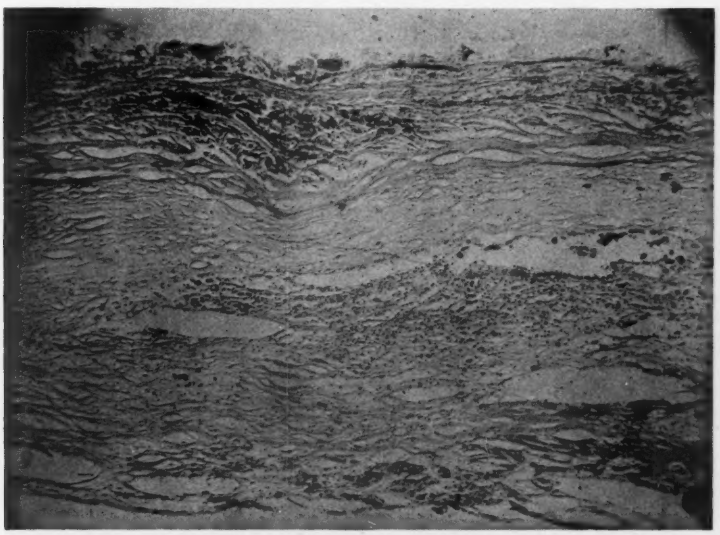

Fig. 5. Photomicrograph. This is characteristic of most of the tissue removed. It consists of fibrous tissue with infiltration of lymphocytes and leukocytes.

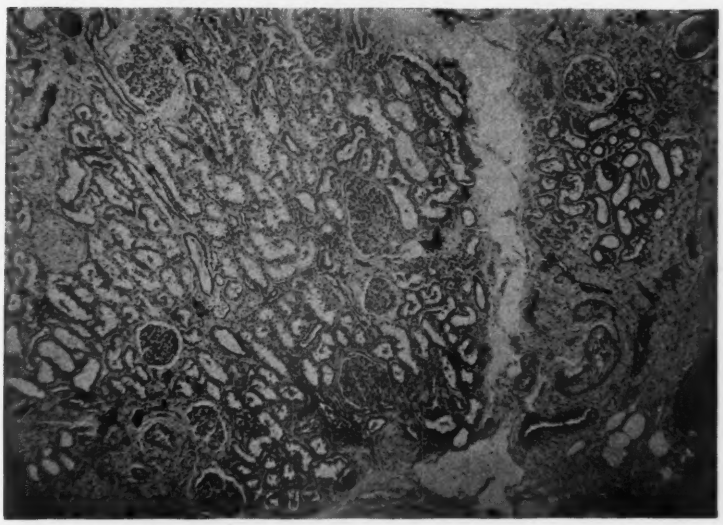

Fig. 6. Photomicrograph of tissue removed. A very small area of adult kidney tissue with marked destruction of glomeruli and dilatation of tubules and scar tissue replacement.

In favor of aplasia of the kidney were: (1) the presence of a bicornuate uterus; (2) absence of the left ureteral orifice and smooth deformity of the left side of the bladder; (3) absence of function from the left kidney. The diagnosis of aplasia was made and confirmed by operation and pathologic examination.

Injection of air into the renal fossa was used as an additional aid in diagnosis. This procedure carries with it the danger of air embolism and is open to criticism on that ground.

The patient has experienced no pain since operation. It is difficult to explain this happy circumstance by nephrectomy. It is also too early after operation to be sure that the pain will not recur. In the meanwhile we have dilated the right ureter.

KIRSCHNER WIRE EXTENSION FOR TREATMENT OF OVERRIDING FRACTURES OF THE CLAVICLE*

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THE correction of overriding fractures of the clavicle not infrequently present a problem of some difficulty. Numerous methods have been contrived to overcome this deformity. Most of these methods aim to pull the point of the shoulder outward, upward and slightly backward in order to reëstablish the normal distance between the sternoclavicular and the acromioclavicular joints. At times the closed methods commonly used are unsuccessful. It is then necessary to resort to open reduction, which, on account of the resultant scar, is sometimes undesirable from a cosmetic standpoint; therefore a method which overcomes for the most part any obvious scarring should be of distinct service.

Inasmuch as the clavicle acts to maintain the distance of the shoulder from the trunk, it is obvious that when fractured, the entire shoulder girdle is apt to fall medially forward and downward, carrying with it the outer fragment of the clavicle, and in case of a complete fracture which becomes disengaged the two fragments override. To secure reposition, therefore, it is necessary that the normal position of the shoulder be reëstablished. The mechanism of reposition must be one which pulls the shoulder and concomitantly the

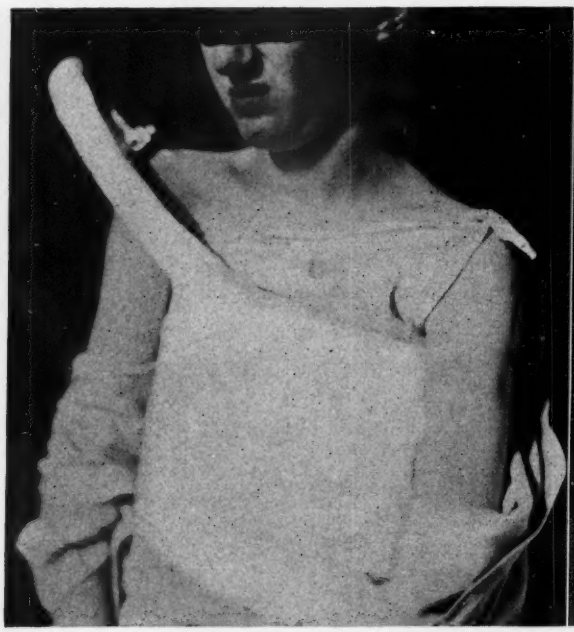

FIG. 1. Front view of patient with traction arch jacket. The action of the elastic pull upon the clavicle can be seen by the manner in which the shoulder is elevated and by the accentuation of the clavicle which is drawn into relief along its entire subcutaneous length.

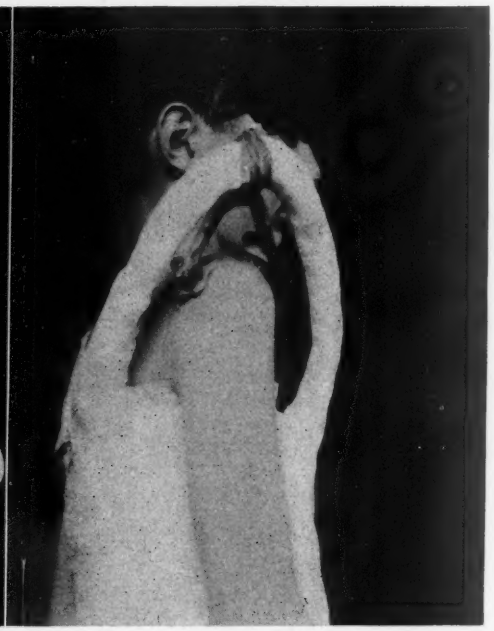

FIG. 2. Lateral view, showing direction taken by the Kirschner wire as can be seen by the diagonal position of the Kirschner spreader. Note position of the plaster arch. The arm is entirely free and can be used for all normal activities.

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outer end of the clavicle, outward, upward and slightly backward. It is a general experience that the maintenance of such a position by any piece of ambulatory apparatus is most difficult, and for this reason the application of direct skeletal traction was considered as a desirable addition to the methods of treatment already in use.

In selecting the site of application of this traction, an easily accessible bony point removed from any important contiguous structures had to be chosen. The prominence finally taken was the acromion process of the scapula. The clavicle is attached to the acromion process by means of extremely strong ligaments through an articulation which permits the direct transmission to the clavicle of any traction force applied to the acromion. The insertion of a wire through the acromion is relatively simple since that bone can be palpated in a subcutaneous position throughout its entire extent. The wire should be placed through a portion of the bone which will bear the stress without danger of having the wire tear out. The acromion is a rather broad and thin process. The superior subcutaneous surface is directed upward dorsally and somewhat laterally, while the inferior surface overhangs the shoulder joint. A wire which penetrates the entire thickness of this bone does not of necessity enter the shoulder joint proper. In order to avoid that articulation, however, it is desirable that a wire should be so inserted as approximately to parallel the plane of the posterior superior surface of the acromion. It is not difficult to insert a thin Kirschner wire through the acromion in this plane under local anesthesia, and a wire so inserted has a firm hold upon the shoulder girdle.

In introducing the Kirschner wire, the spine of the scapula should be palpated until the expansion which forms the subcutaneous surface of the acromion is reached. A site is selected ¾ inch medial to the tip of the process. At this point the Kirschner wire is inserted in an upward and forward direction, approximately paralleling the posterior superior surface of the bone,

Fig. 3. Close-up view, showing the distance which has been maintained between the Kirschner spreader and the plaster arch to permit the application of rubber band traction. The rubber bands span the space between the Kirschner spreader and the arch.

traversing the acromion from its posterior to its anterior edge. Inasmuch as the side of the wire is of necessity held almost against the skin below the acromion, it will be found helpful to depress the skin away from the wire by means of a sterile flat spatula during the process of introduction. With the assistance of this spatula, it is extremely easy to introduce a wire in an upward and forward direction which penetrates the bone and comes out at the anterior aspect of the acromion. Using this directional approach, it is impossible to injure any articular structure associated with the shoulder joint.

After the wire has been inserted and the entrance and exit wounds properly protected, a small Kirschner spreader is attached and proper tension of the wire established. The spreader should be of a height just adequate to span the rounded tip of the shoulder without contacting the skin. A spreader with any greater height

than this adds to the cumbersome nature of the subsequent dressing.

A plaster of Paris jacket is then applied over adequate padding. This jacket must extend from the iliac crest, to which it should be well moulded, and should reach comfortably high into the axillae. Using thin wood veneer strips as a basis, a plaster of Paris arch is articulated to the jacket in such a manner that it spans the point of the shoulder on the affected side, leaving a space adequate to clear the Kirschner stirrup and provide sufficient room to permit elastic extension to be applied to the stirrup. This arch should so clear the shoulder as to enable the surgeon to apply the traction in the indicated direction, that is outward, upward and slightly backward.

After the plaster jacket has been applied, a strip of wood veneer which has been previously soaked in steaming hot water to secure flexibility, is cut to such a length that when arched over the shoulder from the anterior aspect of the jacket to its posterior aspect, it will provide the desired clearance of the stirrup. The strip may be made of multiple layers of the veneer and should be about 1 inch in width. A wrapping of plaster of Paris bandages is applied over the veneer strip, and it is then anchored to the jacket in the proper position with plaster bandages. These are also used to build up the thickness of the arch to a strength adequate to survive any possible stresses it may encounter during the period of treatment. A study of the

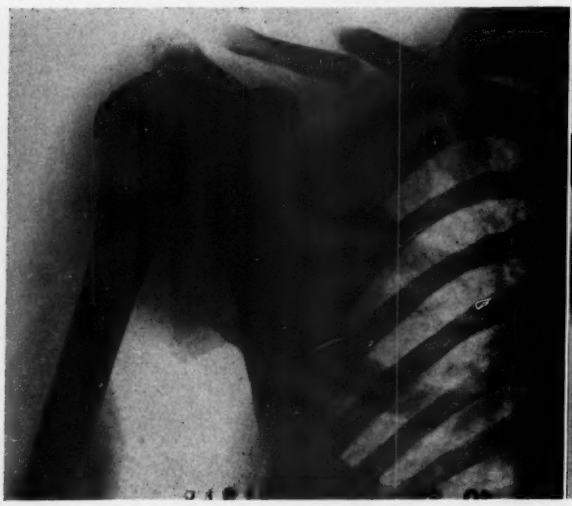

Fig. 4. Roentgenogram showing overriding of the fracture prior to application of extension.

Fig. 5. Kirschner wire in place and correction of overriding under elastic traction.

Fig. 6. Result eight months after injury.

illustrations will help to clarify the construction of this arch.

Traction is applied between the arch and the Kirschner spreader by means of rubber bands. The number of rubber bands required will vary of course with the thickness of the bands, the amount of tension applied to each band, and with the size, weight, and muscular development of the patient. The precise amount of traction can be determined by the reduction of the overriding as judged from digital inspection of the fracture, fluoroscopic examination and *x*-ray films. This type of traction was found to be entirely adequate and sufficiently constant to maintain the normal length of the clavicle throughout treatment.

CASE REPORT

E. M., age 17, was admitted to Lenox Hill Hospital February 10th, 1938 with a fracture of the right clavicle. He had fallen on the street, striking the ground with the right shoulder, but did not come to the hospital for treatment until two days later. During the intervening period he was without medical attention. Due to birth injury, the patient had always been mentally backward.

Pain and disability of the right shoulder were present and this shoulder was about 2 cm. below the level of the left and more medially placed. The right clavicle presented a palpable irregularity at about its mid-portion with obvious deformity. This area was tender to pressure, and *x*-rays revealed a fracture of the middle third of the clavicle with the medial fragment overriding the lateral fragment approximately 1 inch.

On account of the mental status of this patient and the obvious impossibility of preventing him from meddling with any apparatus which might be applied, we decided to attempt reduction by means of skeletal traction. This was done in the manner above described. After the Kirschner spreader had been fastened with adequate tension, elastic traction was instituted by means of rubber bands exerting traction upon the clavicle in an upward, outward and slightly backward direction.

The patient returned from the operating room in a wheelchair. He had no discomfort and was ambulatory in the ward, but his mental condition made him unruly and difficult to discipline. On February 14 the fracture site was no longer palpable as an irregularity along the continuity of the clavicle. However, the patient insisted on playing with the apparatus and introduced a superficial infection at the posterior site of the entrance of the wire which necessitated its removal on February 22. At this time union had obviously progressed to a sufficient degree to prevent any recurrence of displacement. No false motion was detected, and the patient was discharged on February 25, wearing a dressing over the puncture wounds which had already healed. He was seen subsequently on two occasions in the out-patient department. On October 4, 1938 the shoulders were symmetrical as judged by height and distance of the acromion tips from the sternal notch. There was no deformity of the clavicle evident on inspection. *X*-rays showed firm union with a fusiform callus and preservation of the normal clavicular curve without overriding. The scars of the Kirschner wire wounds are insignificant and scarcely noticeable.

COMMENT

This is admittedly a report of a single case, but is presented in order to demonstrate the feasibility of this approach to the solution of the problem of correcting overriding in fractures of the clavicle. The mechanics of the method are simple and applicable by any surgeon who has the facilities for the introduction of a Kirschner wire.

The results in this first case might have been slightly better had we been able to maintain traction over a longer period of time. Inasmuch as the patient was a mental defective and evidently was himself the cause of the superficial infection which ensued, we feel that this complication is one which is not to be anticipated in the average case. The infection subsided immediately upon withdrawal of the wire.

From the experience gained in this case, we feel justified in recommending the method of skeletal traction in cases of overriding fracture of the clavicle, in which it is deemed desirable to overcome the cosmetic blemish of the clavicular irregularity without adding the equally undesirable blemish of an operative scar.

OSTEOGENIC SARCOMA OF THE THYROID GLAND*

REPORT OF A CASE

MARTIN BATTS, JR., M.D.

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OSTEOGENIC sarcoma of the thyroid is an extremely rare lesion. In 1935 Broders and Pemberton reported one case which was the first of its kind observed in the course of a pathologic examination of approximately 40,000 thyroid glands. A survey of the literature at that time revealed only six previously reported cases by Foerster, Pick, Funkenstein, Chavannay and Nadal-Pierre, and Solaro. The following is a report of an additional case.

CASE REPORT

A. J., a 71-year-old female, entered the University Hospital on November 21, 1933 with the complaint of a swelling in the neck. At the age of 19 years the patient had noted a nodule on the left side of the neck, which was not painful and not tender. At the age of 45 years this nodule began to increase in size, and at the time of admission was approximately the size of a baseball. During the six months previous to admission the patient had noted nervousness, anorexia, increased perspiration, palpitation, frequent choking sensations, dyspnea on exertion, and swelling of the ankles. She had lost 15 pounds in weight in six months.

Physical examination revealed a senile, white female, not acutely ill, but showing evidence of recent weight loss. There was complete blindness of the left eye, enophthalmos, and an old traumatic scar of the cornea and iris. The mouth was edentulous. There was a large, nodular tumor 7 cm. in diameter, which seemed to involve the entire thyroid. This tumor was hard in consistency, but there were scattered areas which felt cystic. The trachea was not palpable. There was no bruit or thrill over the tumor. The heart was moderately enlarged and there was an occasional extrasystole. There were a few crackling râles at the right lung base. There was evidence of generalized peripheral sclerosis. The blood pressure was 188/96. There was slight pitting edema of the ankles.

Examination of the blood revealed a hemoglobin of 68 per cent, a red blood cell count of 4,100,000 per cu. mm., and a white blood cell count of 7,850 per cu. mm. The Kahn test for syphilis was negative. The urine examination was negative. *X*-ray examination of the chest revealed a large, round mass in the base of the neck compressing the trachea and reducing its lumen to about one-quarter of its former width; scattered calcium deposits were noted throughout the anterior position of the tumor. The basal metabolic rate was plus 31 per cent, with a pulse rate of 80 per minute. The electrocardiogram was normal. Laryngoscopic examination was negative.

A provisional diagnosis of adenomatous goiter with hyperthyroidism and questionable malignant degeneration was made. On November 29, a subtotal thyroidectomy was done by Dr. F. A. Coller under local novocaine anesthesia. The pathological diagnosis rendered by Dr. C. V. Weller was: "This is a very unusual neoplasm. It is a supporting tissue tumor which forms osteoid tissue and bone, but which is arising in an old thyroid adenoma with remains of the old glandular structures still present. The neoplasm can be designated a spindle-celled osteoid-osteo-fibrosarcoma. Some areas show giant cells of the type found in benign bone tumors. Early metastases from this neoplasm are not anticipated."

The immediate postoperative condition was good. Preoperatively the patient had been placed on Lugol's solution, which was continued after operation. On the first postoperative day there was a fever of 102 degrees. Thereafter the patient continued to run an afternoon fever which remained unexplained until the eighth postoperative day when fluctuation was noted in the left angle of the wound. Incision was done, and drainage of seropurulent material was carried out, after which

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the wound healed. The patient was discharged on the eighteenth postoperative day with instructions to continue with the Lugol's solution.

Fig. 1.

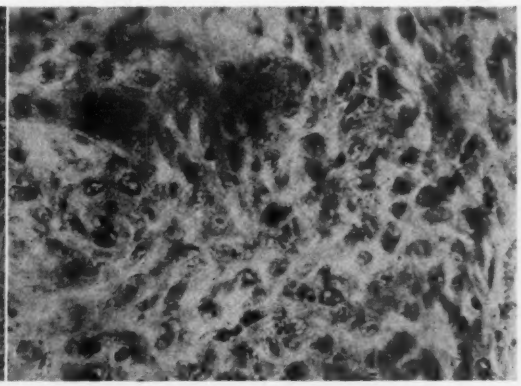

Fig. 2.

Following discharge the patient was treated with heavy doses of *x*-ray radiation. She continued to show signs and symptoms of myocardial insufficiency, and on March 23, 1934 died of cardiac failure. An autopsy was not obtained.

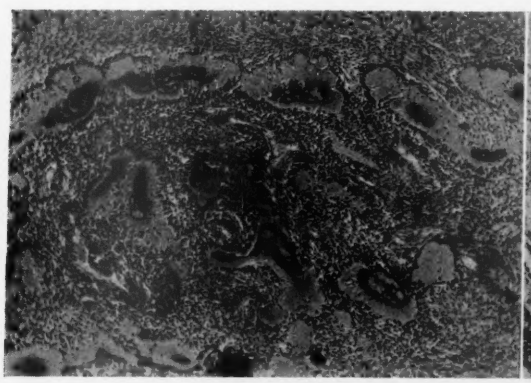

Fig. 3.

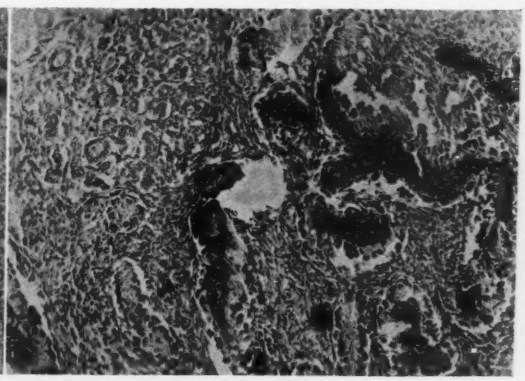

Fig. 4.

To account for the occurrence of an osteogenic sarcoma in such an unusual location, Broders and Pemberton decided that the sarcoma arose from the stroma of the thyroid gland by dedifferentiation, or anaplasia, of fibroblasts. This was substantially the same concept held by Foerster and Solaro. They further believed that the bone was a direct descendant of sarcomatous tissue, as held by Pick, Funkinstein, and Solaro; the sarcoma cells produced sarcomatous osteoblasts, which in turn produced osteoblasts that were indistinguishable from those of benign osteoblasts, followed by the formation of osteoid tissue and true bone.

The above explanation is in full accord with the cellular or classical theory of bone formation, which attributes to the osteoblast the important rôle in the formation of bone. In this connection it is interesting to note the newer theories of bone formation. Leriche and Policard expressed the view that formation of bone is the result of a metaplasic change in the connective fundamental substance; this osseous metaplasia can occur in all types of connective tissue; the cells themselves do not play the part classically attributed to them, that is to say, osteoblasts do not secrete true osseous substance between the cells; rather, the osseous transformation of connective tissue is a phenomenon independent of all cellular action, and is an interstitial and humoral process. The work of Huggins in which heterotopic ossification was produced by

transplants of the epithelium of the urinary bladder in the abdominal wall seems to support the latter theory. Perhaps this theory also offers the best explanation for the occurrence of an osteogenic sarcoma of the thyroid gland.

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As a rule, a sarcoma of the stomach, where its surface impinges on the lumen, shows an excavated ulcer with undermined edges. Actually many cases of sarcoma of the stomach have been diagnosed under *x*-rays as chronic ulcer.

From—"Surgery of the Alimentary Tract" by Devine (Williams & Wilkins Company).

PHLEGMONOUS PROCTITIS

REPORT OF A CASE

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THE usual rectal complications following pregnancy are hemorrhoids of all kinds, fissures, abscesses; the more serious difficulties are complete tears, especially those with destruction of part of the sphincter mechanism. Several cases of rupture of the rectum following labor have been reported, all of these occurring in patients who had a preëxisting stricture of the rectum.

Phlegmonous proctitis can be brought about by the introduction of chemical irritants into the rectum, or can be produced by a hot enema, or a long protracted hard labor. These are probably etiologic factors in the present case. The patient was given paraldehyde per rectum for analgesic purposes at the onset of labor; several hours later it was repeated. On the tenth day postpartum the patient was given a soapsuds enema, and, at that time, complained that it felt warm; the day following she complained of pain in the coccygeal region which continued; there was no rise in temperature, no diarrhea. On the fourteenth day she returned home and on that evening began to show symptoms of a severe proctitis. In view of the fact that the symptoms came on fourteen days after the introduction of paraldehyde and fourteen days after labor, and almost immediately after the hot enema, it is the opinion of the author that the latter was the most likely etiologic factor.

CASE REPORT

Mrs. L. C., aged 28, primipara, reported a negative past history, with the exception of childhood diseases. She had been given some injections for hemorrhoids in 1934.

Labor started at 1:00 A.M. on November 5, 1935, and ended at 5:24 P.M. that same day. For analgesia she was given a hypodermic injection of $\frac{1}{3}$ gr. pantopon and $\frac{1}{200}$ gr. scopolamine; also 3 gr. sodium amytal and nembutal. At 10:00 A.M she was given 4 drams paraldehyde in 3 ounces of olive oil, and this was repeated at 12:45 P.M. At 5:24 P.M. she was delivered after median episiotomy and application of low forceps, nitrous oxide and ether being given by inhalation.

The postpartum course was normal; on November 7, 1935, her bowels moved; she was troubled by external hemorrhoids to which nupercaine ointment was applied daily. On November 12, 1935, she was given a soapsuds enema which produced fair results. On November 13, 1935, her bowels moved, but her hemorrhoids gave her considerable pain which was somewhat alleviated by nupercaine ointment. On the morning of November 14, 1935, she was given a soapsuds enema preparatory to surgery for cervical repair; at the time of the enema she reported to her obstetrician that the fluid felt rather warm. On November 15, 1935, she began to complain of pain in the region of the coccyx, and for this the infra-red ray was applied. On November 16, 1935, she was given a soapsuds enema with excellent results, and again the infra-red was applied to the lower back. On November 17, 1935, she had a normal bowel movement and a hot water bag was placed over the region of the coccyx to relieve her pain. On November 18, 1935, the patient felt quite well, and was taken home later in the day.

After returning home the patient had several bowel movements, accompanied by a purulent discharge and some pain. The author first saw the patient that evening at her home and found her rather nervous and upset and in bed. A large piece of sloughing mucous membrane, at least 2 inches in width and 6 inches in length was protruding from the anus; further examination revealed more of this mucous membrane slough within the anal canal and rectum. The slough protruding from the anus was excised. On the morning of November 19, 1935, there

was another equally large piece of sloughing mucous membrane protruding from the anus, and this was excised. During the day she passed another slough 5 inches in length and 2 inches in width; that evening a speculum was introduced, and an extremely raw surface was seen in the upper part of the rectum. Further examination was not deemed advisable at this time. Temperature and pulse were normal all day.

On November 20, 1935, the rectum was examined and found to be very raw and denuded of its mucous membrane; there was some purulent discharge from the anus. Two ounces of 1½ per cent mercurochrome were instilled into the rectum and the patient was told to take one ounce of mineral oil three times daily. On November 21, 1935, there were two small bowel movements with some purulent discharge. The patient complained of her hemorrhoids, but, due to the severity of her proctitis, nothing was done for this for the time being.

On November 22, 1935, the patient stated she had a soft liquid bowel movement. Examination revealed very little purulent discharge, but the entire rectum was raw and denuded. On November 24, 1935, patient had a very good day, with the exception of some rectal bleeding. The following day she was quite well, had a bowel movement with no bleeding, and then passed several clots and some fresh blood. At 5:00 P.M. she was still bleeding and examination revealed the rectum filled with clots which were removed. Bleeding was checked by packing the rectum, especially the raw bleeding surface, with vaseline gauze. There being no further bleeding, the packing was removed the next day. From then on progress was very good; the wound began to heal and the patient began to feel better; she was given 2 ounces of mineral oil per rectum every night as a retention enema. A high caloric, low residue diet produced rather small liquid bowel movements.

Due to the acuteness and severity of her condition the patient was not sigmoidoscoped until December 4, 1935, at which time examination revealed that a stricture of the rectum had developed 10 cm. above the anus, through which the small ⅜ inch sigmoidoscope could not be passed. Dilatation of the rectum was begun December 5, 1935, Hegar dilators being used. The No. 8 dilator was used that day and dilatation was repeated every fourth day.

On June 3, 1936, a No. 22 dilator was passed and at the same examination a ¾ inch sigmoidoscope was passed beyond the stricture area which was about 1 inch in length and completely encircled the bowel wall. Much granulation tissue could be seen in the stricture area, which bled rather easily. Dilatation was continued every fourth day and the patient continued to improve. Bowel function was quite normal, with mineral oil being used as a lubricant.

On June 20, 1936, a barium enema showed a definite constriction at the midpoint of the rectum, which, however, was regular and did not bar easy access of the opaque meal; there was some lack of haustration in the descending colon.

Gradually dilatation was brought about, and on December 28, 1936, the large No. 22 dilator was used with no difficulty; the sigmoidoscope was easily passed and no pathology was seen with the exception of a slight constriction at the site of injury. The patient thereafter remained well.

DISCUSSION

According to Frederick Wallis[1] one of the causes of stricture of the rectum may be attributed to a long protracted labor in which the child's head becomes jammed in the pelvis for some hours. By direct pressure it may cause ultimate destruction of an area of mucous membrane, which generally separates, and, due to constant exposure to fecal material, becomes septic. This may be the origin of progressive ulceration and stricture formation. However, if this were true, many cases of rectal ulceration and stricture formation would be seen following these long protracted labors. The rectum is movable and can be easily accomodated along the sides of the sacral promontory in an already crowded pelvis. Further, in the case under discussion, the patient, a primipara, was in labor for sixteen hours which is, according to Williams,[2] two hours less than the normal for a primipara. Further, in view of the fact that the symptoms in this case did not come on until fourteen days following

delivery, this etiologic factor can be eliminated.

The author knows of two cases in which there was a pronounced local irritation and generalized rash following the application of nupercaine to the anal region. In this case the drug was used to allay the soreness of hemorrhoids following delivery, but at no time were there any signs of local irritation.

Paraldehyde has been used per rectum for analgesic purposes with no harmful effects either locally or generally. Kane and Roth[3] report ninety-one cases with no ill effects; Rosenfield[4] used it in 400 cases with no harm; Parker[5] has used it effectively with no bad results and reports a case in which a patient received five rectal injections, one every five hours and forty-five minutes, with no injury to the patient; Colvin and Bartholomew[6] report one hundred cases with no ill effects; Kane and Roth[7] in 1936 reported a group of cases with no ill effect of heart, kidney, lungs, liver, respiratory center and bowel. Further, the onset of symptoms fourteen days following the use of paraldehyde makes the author believe that this drug played no part in the production of the symptoms.

In view of the fact that the patient complained of an enema being too hot, and in view of the fact that the symptoms (pain over the region of the coccyx, tenesmus and diarrhea) arose four days later when sloughing began, it is the opinion of the author that the hot enema, given on the tenth day, preparatory to surgery, was the primary etiologic factor in this case. A soapsuds enema in itself is quite irritating to the bowel, as evidenced by the great amount of mucus that is excreted by the bowel following it. It is the opinion of the author that the pathology in this case which brought about a sloughing of the mucous membrane of the rectum can be compared to that of a third degree burn. At a meeting of the North of England Obstetrical and Gynecological Society,[8] several cases were reported of more extensive rectal injury following the use of hot enemas postpartum, and Mr. Kelly, in particular, reported a case in which there was a sloughing of the mucous membrane of the rectum and vaginal wall and the prolapse of gangrenous loops of tissue out of the cavity, closely resembling coils of small gut.

SUMMARY

A case of phlegmonous proctitis apparently brought on by a warm enema has here been presented. Due to the great amount of sloughing stricture formation followed. With very conservative treatment, after the acuteness of the condition had subsided, a good curative result was obtained, with almost complete dilatation of the bowel and well functioning rectosigmoid.

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NEW INSTRUMENTS

AN INTESTINAL HOLDING CLAMP*

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IT is frequently desirable to elevate a segment of the intestinal tract. The usual plan is to use a clamp with

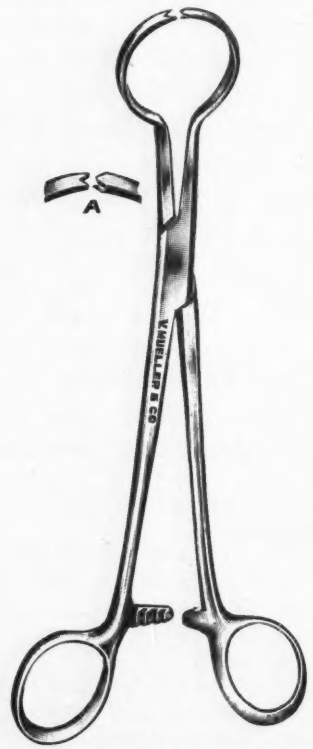

FIG. 1. Ring clamp for holding a segment of intestine.

fenestrated or rubber covered blades. The application of these clamps is a maneuver that requires a good deal of judgment, for, if too much pressure is exerted, some damage of the intestinal wall will occur, and if too little, the instrument will slip.

The clamp here presented and illustrated has been found useful in overcoming these difficulties. It is simply an adaptation of the principle of the ring clamp. This ring has a diameter of 2.7 cm. which is of sufficient size to encircle the colon. The jaws are of the interlocking, rat-toothed type. The over-all length of the clamp is 19.5 cm.

Method of Use. The mesentery of the segment of intestine which the surgeon desires to control is pierced in an avascular area at the bowel margin with a pointed instrument such as the tip of a Kelley artery clamp. The ring clamp is then applied in such a manner as to encircle the bowel, perhaps best by slipping a fingertip through the opening just made by the artery clamp through the mesentery, placing a tip of the opened clamp jaw against the glove; the finger is slowly withdrawn with the tip of the clamp blade following until all danger of scraping the gut is over. The clamp is then closed. The jaws of the clamp meet and interlock through the mesenteric opening. Adequate control of this isolated area of intestine is now possible. There are many situations in intestinal surgery where this clamp has been found exceedingly useful, but its chief value is in control of the mobilized duodenum or pyloric end of the stomach before and during application of crushing clamps prior to section of this organ in gastric resection.

The utility of this clamp is confined to its holding properties, for it does not prevent the passage of intestinal contents.

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A NEW TYPE OF TISSUE FORCEPS*

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THE usual type of tissue forceps with teeth across the end often will not grasp easily tags of tissue, and, if the piece happens to be bone or cartilage, it will invariably slip out of the teeth. When repairing cleft palates or doing other work in the mouth, this is especially annoying. The forceps illustrated was designed to grasp these small bits of tissue.

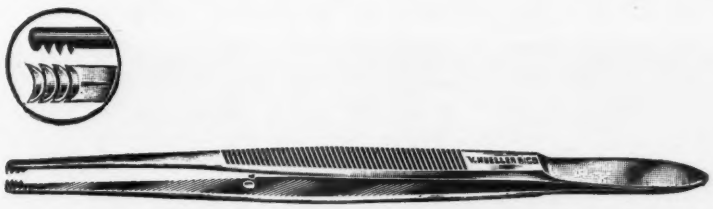

FIG. 1. Tissue forceps with multiple teeth running up the shank instead of across the end. A wider area of tissue is grasped and held more securely with less pressure necessary on each tooth and, therefore, less trauma to the tissue. The round insert does not depict the small individual teeth as clearly as they are on the forceps. (*Arch. Surg.*, June, 1940.)

Its essential distinguishing feature is that the teeth run up the shank rather than across the end; there are three small interlocking teeth instead of the usual two on one side and one on the other. A wider area may be grasped and held more firmly, but with less pressure and thereby less trauma by the individual teeth. Another advantage is that in work in cavities where the grasping of a needle is difficult, this forceps will grasp and steady the needle until a locked forceps can be put on it for removal; if the tissue is soft, the forceps is all that is necessary for withdrawal.

After using the forceps originally in the mouth with satisfaction, they were found to be helpful in other work and many times in the approximation of deep tissues and of skin; they have also been used in gynecological work with some enthusiasm.

Since the teeth run up the forceps, the grasp is somewhat sideways, and, for picking up edges at right angle or perpendicular to the incision, the small end tooth forceps may be preferable.

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NEW RECTAL SCISSORS*

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IN the practice of proctology, in both hospital and office, the use of a scissors through an anoscope is often required. The most common instance is in the post-operative care following surgery of the rectum.

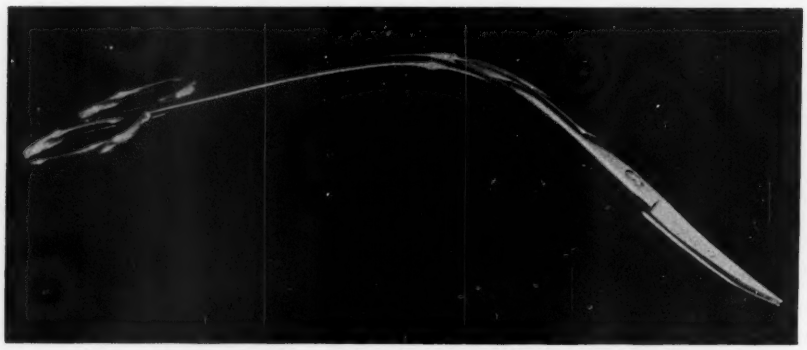
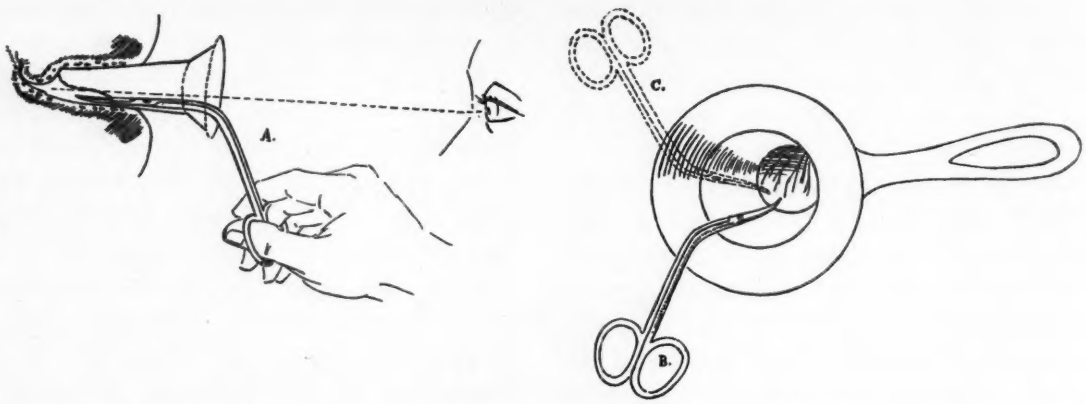


FIG. 1. Lateral view of scissors showing approximate 45 degree angle near middle of shaft.



A B AND C

FIG. 2. A, showing the scissors inserted through the anoscope in cutting position and an unobstructed view. B and C, showing the scissors inserted through anoscope in other cutting positions and unobstructed views.

Ten days to two weeks after operation observation through an anoscope frequently reveals varying amounts of excess granulation tissue which must be removed for proper and smooth healing. Varying strengths of silver nitrate and other cauterizing solutions are not effective since they merely cauterize the surface so that too frequent applications are required. The action of these various solutions, even when carefully neutralized and when adjacent tissue is protected, cannot be perfectly confined to one area. The liquid spreads and thus causes deleterious effects and much prolonged anal discomfort.

The most effective treatment is excision of the excess tissue. Since no sensory nerves are present, the tissue can be excised pain-

* From the Department of Proctology, College of Medical Evangelists. Read before the Proctologic Staffs of the White Memorial and the Los Angeles County General Hospitals.

lessly by means of a scissors through an anoscope. With a straight or double curved scissors the job is done with difficulty if at all, since the operator's hand and the finger grips of the scissors are constantly in the line of vision. If the operator is successful in manipulating the scissors in such a way that the hand is not in the line of vision he will find that the light directed into the rectum through the anoscope is obstructed by some part of the scissors. Cutting has to be done almost entirely by sense of touch and in the dark.

In an attempt to simplify and facilitate the performance of this technical procedure a new type of rectal scissors* has been devised. The scissors are similar to the conventional type, differing mainly in the construction of the handle which forms an angle of approximately 45 degrees near the middle of the shaft. (Fig. 1.) When the blades of the scissors are inserted through the scope and placed in the cutting position, the finger grips and the operator's hand are not in the line of vision and no part of the scissors blocks the path of light. Thus an unobstructed view with proper lighting is obtained no matter in which quadrant of the canal the procedure is to be carried out. Figure 2 shows the scissors inserted through the anoscope with the blades in the cutting position and an unobstructed view.

Other changes from the ordinary rectal scissors have been made: the tips of the cutting blades are not sharply pointed, but are barely blunted so that if the points accidentally touch adjacent tissue they will not puncture, but will slide off. Yet the tip is sufficiently pointed so that it can be used for cutting; the cutting edges of the blades are serrated so that the tissue will not slip out when the blades are closed. (The serrated edge is used on several other types of scissors now on the market.) The curve of the cutting blades is far enough from a straight line so that there is just sufficient curve to conform with the slight angle of the tissues. Thus the tips of the blades can cut without great elevation of the handle.

The angled handle and the very slight curve of the blades of the scissors allow for convenience and relative ease in excising tissue from difficult areas and in deep wounds.

*These scissors are manufactured by J. Sklar Manufacturing Company, Long Island City, N. Y.

BOOK REVIEWS

CLINICAL ROENTGENOLOGY OF THE ALIMENTARY TRACT. By Jacob Buckstein, M.D. Philadelphia, 1940. W. B. Saunders Company. Price $10.00.

The author in his preface says, "Roentgenography has today become so essential a procedure in the diagnosis of lesions of the alimentary tract that a knowledge of its potentialities should well form part of the armamentarium of every practitioner in the field of medicine and surgery." Inasmuch as it has been Dr. Buckstein's privilege for more than twenty years to see (in Bellevue Hospital, New York) a vast amount of material dealing with the alimentary tract from the roentgenologic, operative and postoperative viewpoints, he has presented material as examples of both normal and pathologic conditions for portrayal in the pages of his book. And he has done a tip-top job. The book is well written and understandable; the 525 illustrations are clear and really illustrate, and there is a bibliographical and subject index. Surgeon, gastroenterologist and internist will find this work interesting and instructive.

ORTHOPEDIC OPERATIONS. By Arthur Steindler, M.D. Springfield, 1940. Charles C. Thomas. Price $9.00.

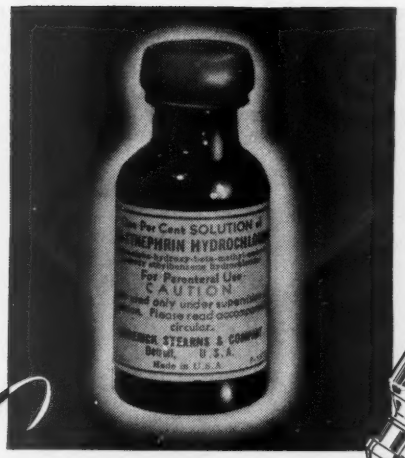
The author of this work needs no introduction to the surgical fraternity of America. He is preeminent in his field, that of orthopedic surgery, and his book echoes or mirrors the knowledge Dr. Steindler has acquired, sifted and evaluated. Therefore, he has written a preeminently practical book, a book that answers such questions as How, When, With What Results? Unlike the usual book on this subject, *Orthopedic Operations* lays special stress upon the indications and end results in specific orthopedic conditions. Much information (bad procedures have not been introduced) has been put into its 766 pages, which also contain 865 illustrations on 322 figures, chapter bibliographies, 1756 subject index and 1469 author index references. We agree with the statement, "Steindler's new book guides the operator quickly to the exact practical procedure which is best fitted to meet each precise clinical situation."

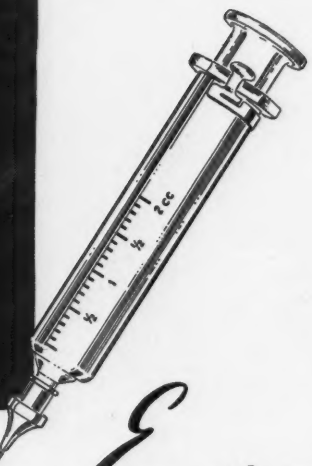
TREATMENT OF WAR WOUNDS AND FRACTURES WITH SPECIAL REFERENCE TO THE CLOSED METHOD AS USED IN THE WAR IN SPAIN. By J. Trueta, M.D. With a Foreword by H. Winnett Orr, M.D. New York, 1940. Paul B. Hoeber, Inc. Price $2.50.

The author tells us that his work is "an amplification of a small manual which was published during the war in Spain, first in Catalan and later in Spanish. The original text was written under war conditions." We are told that open fractures produced in road accidents or in industry do not differ essentially from those produced by aerial bombs or falling masonry. Also, that the fundamental principles of treatment are the same whatever the cause. Dr. Orr, in his Foreword states, "It has been demonstrated now by many surgeons that drainage, skeletal fixation in plaster of Paris casts, and protection against the trauma and re-infection caused by frequent dressings will enable a high percentage of these patients to recover. Dr. Trueta has brought this lesson home to us by a most convincing demonstration under military conditions." This tells what this small book of 146 pages covers. There are 48 illustrations, a bibliography and an index. Unless familiar in every detail with this method we urge every surgeon, especially those dealing with fractures and traumatic injuries, to possess this book.

A TEXT-BOOK OF GYNAECOLOGY. By James Young, M.D. Fifth Edition, Revised. London, 1939. Adam & Charles Black. Price $5.00.

A Text-Book of Gynaecology first appeared in 1921 and now is offered in its fifth edition. This speaks for itself. Although it has been revised and certain parts rewritten, this edition differs from the former in having added the important contribution of sex endocrinology, a consideration of the synthetic estrogens and the appraisement of their clinical value by The Therapeutic Trials Committee of the Medical Research Council. For anyone who wishes to get or keep abreast of modern gynecological teaching this work, containing 425 pages, and 226 illustrations, is recommended.





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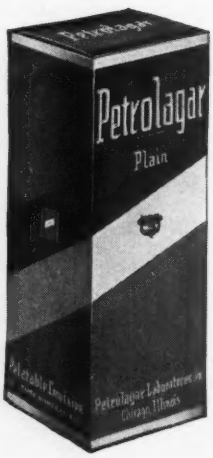

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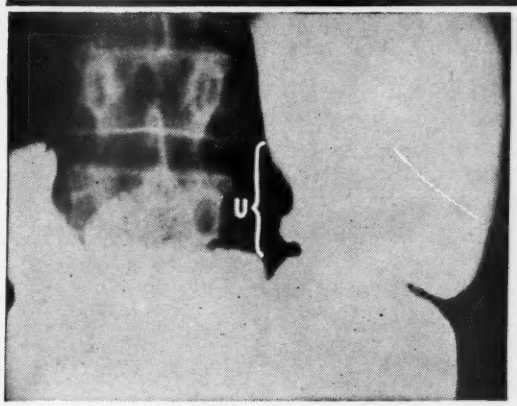

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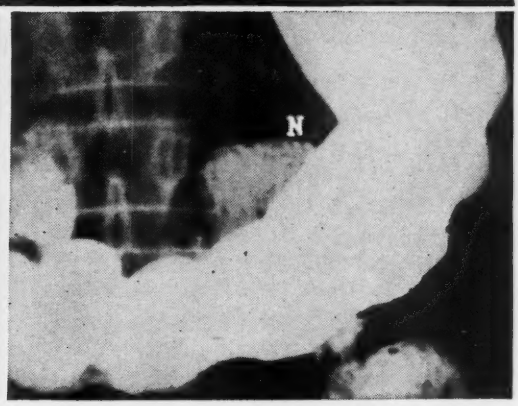

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SOCIETY MEETINGS

AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS
Boston, January 21–25, 1940. (Secy.—Dr. Carl Badgley, University Hospital, Ann Arbor, Mich.)

AMERICAN ASSOCIATION FOR THE STUDY OF GOITER
Rochester, Minn., April 15–17, 1940. (Secy.—Dr. W. Blair Mosser, Kane, Penna.)

AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA
Atlantic City, June 7–8, 1940. (Secy.—Dr. Ralph G. Carothers, 409 Broadway, Cincinnati)

AMERICAN ASSOCIATION OF GENITO-URINARY SURGEONS
Skytop, Penna., June 20–22, 1940. (Secy.—Dr. Charles C. Higgins, 2020 East 93rd Street, Cleveland)

AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS
New York, June 4–7, 1940. (Secy.—Armour G. Park, 540 N. Michigan Ave. Chicago)

AMERICAN ASSOCIATION OF OBSTETRICIANS, GYNECOLOGISTS AND ABDOMINAL SURGEONS
Excelsior Springs, Mo., September 19–21, 1940. (Secy.—Dr. James R. Bloss, 418—11th St., Huntington, W. Va.

AMERICAN ASSOCIATION OF RAILWAY SURGEONS
Chicago, September 16–18, 1940. (Secy.—Dr. D. B. Moss, 547 W. Jackson Blvd., Chicago)

AMERICAN COLLEGE OF SURGEONS
Chicago, October 21–25, 1940. (Secy.—Dr. Frederic A. Besley, 215 N. Sheridan Rd., Waukegan, Ill.)

AMERICAN CONGRESS OF PHYSICAL THERAPY
Cleveland, September 2–6, 1940. (Secy.—Dr. Richard Kovacs, 2 East 88th Street, New York)

AMERICAN GYNECOLOGICAL SOCIETY
New York, June 17–19, 1940. (Secy.—Dr. Richard W. TeLinde, 11 East Chase St., Baltimore)

AMERICAN MEDICAL ASSOCIATION
New York, June 10–14, 1940. (Secy.—Dr. Olin West, 535 N. Dearborn Street, Chicago)

AMERICAN ORTHOPAEDIC ASSOCIATION
Kansas City, May 6–9, 1940. (Secy.—Dr. Ralph K. Ghormley, The Mayo Clinic, Rochester, Minn.)

AMERICAN PROCTOLOGIC SOCIETY
Richmond, June 9–11, 1940. (Secy.—Dr. Curtice Rosser, 710 Medical Arts Bldg., Dallas)

THE AMERICAN SOCIETY OF ANESTHETISTS, Inc.
New York, April 10, 1940. (Secy.—Dr. Paul M. Wood, 745 Fifth Avenue, New York)

AMERICAN SURGICAL ASSOCIATION
St. Louis, May 1–3, 1940. (Secy.—Dr. Charles G. Mixter, 319 Longwood Avenue, Boston)

ASSOCIATION OF MILITARY SURGEONS OF THE U. S.
Cleveland, October, 10–12, 1940. (Secy.—Dr. H. L. Gilchrist, Army Medical Museum, Washington, D. C.)

CONGRESS OF ANESTHETISTS
Chicago, October 21–25, 1940. (Secy.—Dr. E. Klaus, 1699 W. 25th Street, Cleveland)

INTERNATIONAL COLLEGE OF SURGEONS
Venice, Fla., February 11–14, 1940. (Secy.—Dr. Charles H. Arnold, 903–15 Terminal Bldg., Lincoln, Nebr.)

INTER-STATE POST-GRADUATE MEDICAL ASSOCIATION OF NORTH AMERICA
Cleveland, October 14–18, 1940. (Secy.—Dr. Tom B. Throckmorton, 406 Sixth Avenue, Des Moines)

NEW ENGLAND SURGICAL SOCIETY
Poland Spring, Me., September 27–28, 1940. (Secy.—Dr. John F. Gile, Hanover, New Hampshire)

PACIFIC COAST SOCIETY OF OBSTETRICS AND GYNECOLOGY
San Francisco, November 6–9, 1940. (Secy.—Dr. T. Floyd Bell, 400—29th Street, Oakland, Calif.)

PACIFIC COAST SURGICAL ASSOCIATION
Portland, April 3–6, 1940. (Secy.—Dr. H. Glenn Bell, University of California Hospital, San Francisco)

PAN-PACIFIC SURGICAL ASSOCIATION
Honolulu, 1942. (Secy.—Dr. Forrest J. Pinkerton, Young Bldg., Honolulu)

SOUTHEASTERN SURGICAL CONGRESS
Birmingham, March 11–13, 1940. (Secy.—Dr. B. T. Beasley, 701 Hurt Bldg., Atlanta)

SOUTHERN SURGICAL ASSOCIATION
Hot Springs, Va., December 10–12, 1940. (Secy.—Dr. Alton Ochsner, 1430 Tulane Avenue, New Orleans

WESTERN SURGICAL ASSOCIATION
Topeka, Kans., December 6–7, 1940. (Secy.—Dr. Albert H. Montgomery, 122 S. Michigan Blvd., Chicago)

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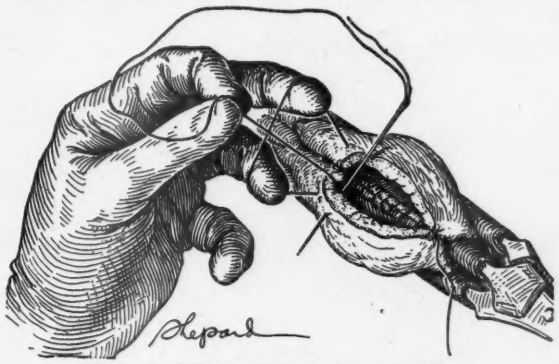

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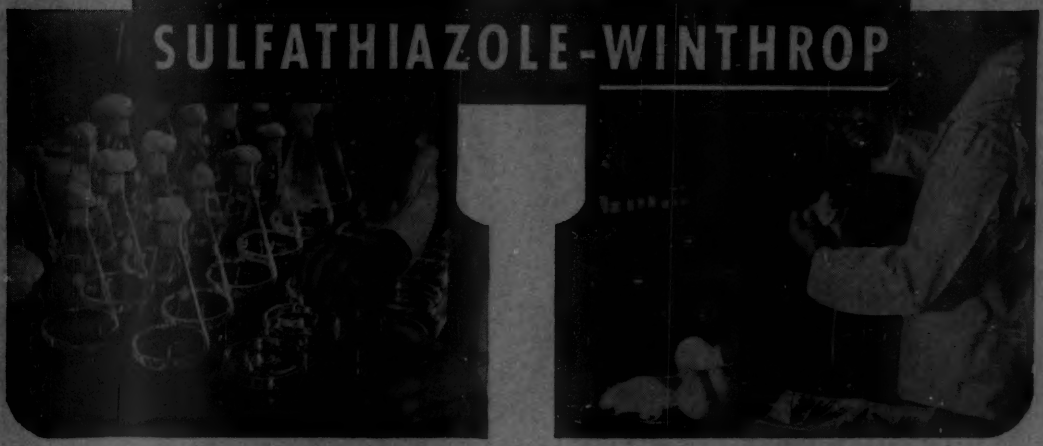

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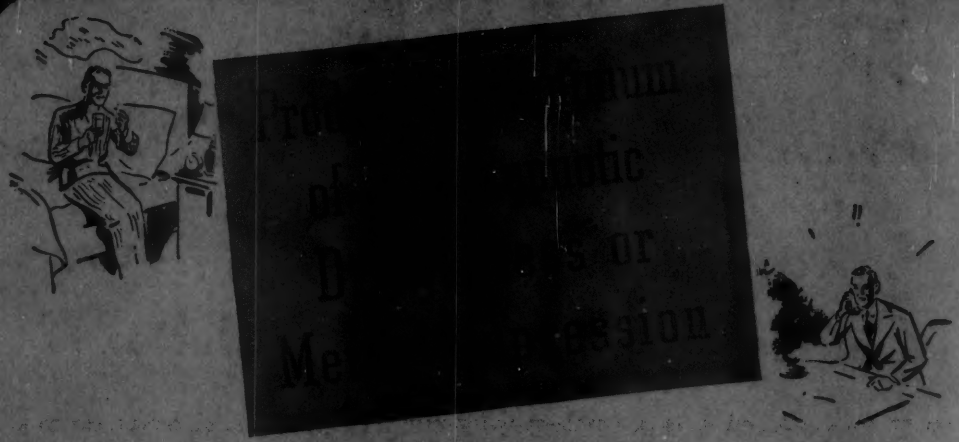

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